STUDY OF EFFECTIVENESS OF DEXAMETHASONE AS AN ADJUVANT TO LOCAL ANESTHETIC MIXTURE IN PROVIDING POST OPERATIVE ANALGESIA IN SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK

Dissertation submitted to

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(Branch – X) ANAESTHESIOLOGY

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CHENNAI

TAMILNADU

DECLARATION

I hereby declare that the dissertation entitled **"Study of effectiveness of dexamethasone as an adjuvant to local anesthetic mixture in providing post operative analgesia in supraclavicular brachial plexus block"** has been prepared by me under the Guidance of **PROF. DR C.R.KANYAKUMARI M.D., D.A.,** Professor and Director, Institute of Anesthesiology and Critical Care, Madras Medical College, Chennai in partial fulfillment of the regulations for the award of the degree of M.D [Anaesthesiology], examination to be held in April 2012.

This study was conducted at Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai.

I have not submitted this dissertation previously to any university for the award of any degree or diploma.

Date :
Place : Chennai **Dr. C.KARTHIKEYAN**

CERTIFICATE

This is to certify that the dissertation entitled, **"Study of effectiveness of dexamethasone as an adjuvant to local anesthetic mixture in providing post operative analgesia in supraclavicular brachial plexus block"** submitted by Dr.C.Karthikeyan in partial fulfilment for the award of the degree of Doctor of Medicine in Anaesthesiology by the Tamilnadu Dr. M.G.R. Medical University, Chennai is a bonafide record of the work done by him in the INSTITUTE OF ANAESTHESIOLOGY & CRITICALCARE, Madras Medical College, during the academic year 2009-2012.

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CONTENTS

INTRODUCTION

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“For all the happiness that mankind can gain
It is not in pleasure but in relief from pain”

- JOHN DYRDEN

“Pain, like pleasure is passion of the soul,
That is an emotion and not one of the senses”

- PLATO AND ARISTOTLE (375 B.C)

Pain is a fundamental biological phenomenon. The International Association for the Study of pain has defined pain as an unpleasant sensory and emotional experience associated with actual or potential tissue damage. Pain is always underestimated and under treated. The relief of pain during surgery is the main part of anaesthesia.

In 1784 James Moore used mechanistic concepts to promote neural compression as a useful technique for the provision surgical anaesthesia.

In 1855 Gadecke (German) isolated an alkaloid from leaves of coca plant. In 1860 Albert Nieemann was successful in isolating and naming the alkaloid from the leaves of erythroxylon coca.

Brachial plexus block is a popular and widely employed regional nerve block technique for perioperative anesthesia and analgesia for

surgery of the upper extremity. Regional nerve block avoids the unwanted effect of the anesthetic drugs used during general anesthesia and the stress of laryngoscopy and tracheal intubation.

Brachial plexus block is approached at the level of trunks and the compact arrangement of trunks at supraclavicular level gives a high success rate with minimal drug volume and a dense blockade. Hence the supraclavicular approach is the method of choice for blocking the brachial plexus.

William steward halsted first performed brachial plexus block in 1895. In 1911 Kulenkampff and Hirshel described the first percutaneous brachial plexus block by supraclavicular and axillary routes respectively.

Of the various techniques the most widely practiced methods are the classical technique described by Patrick, vertical plumb pop technique described by Brown, 1st rib walk over technique described by Bonica and Moore, and the subclavian perivascular technique described by Winne and Colins.

Of the various local anesthetics used for brachial plexus block lignocaine and bupivacaine are used most frequently in our set up.

Adrenaline is added to the local anesthetics to prolong the duration of action and to minimize the systemic absorption.

To prolong the duration of analgesia various drugs have been studied as adjuvants to the local anesthetic solution. These adjuvant drugs are ideally expected to prolong the analgesic effects without causing any systemic side effects or prolonging motor blockade.

HISTORY

1. 1858 - Theory of pain was a separate and distinct sense was definitely formulated by Mortiz S.Schiff.

2. 1884 - William Halsted and Alfred Hall – idea of injecting cocaine into nerve trunk.

3. 1911 - G. Hirschel performed first percutaneous axillary brachial plexus block.

4. 1911 - D.Kulenkampff performed supraclavicular brachial plexus block.

5. 1943 - Lidocaine was synthesized by Lofgreen and Lundquvisit

6. 1956 - Bupivacaine synthesized by Ekenstam

7. 1963 - Bupivacaine introduced clinical practice by Telivuo

8. 1965 - Melzock and Walts propounded the Gate Control Theory of pain.

AIM OF THE STUDY

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Aim of the study is to evaluate the following observations in patients receiving either dexamethasone or the placebo as an adjuvant to local anesthetic mixture in supraclavicular brachial plexus block.

1. Onset of sensory blockade.
2. Onset of motor blockade.
3. Quality of anesthesia.
4. Duration of sensory blockade.
5. Duration of motor blockade.
6. Hemodynamic changes.
7. Complications.

ANATOMY OF THE BRACHIAL PLEXUS

THE BRACHIAL PLEXUS:

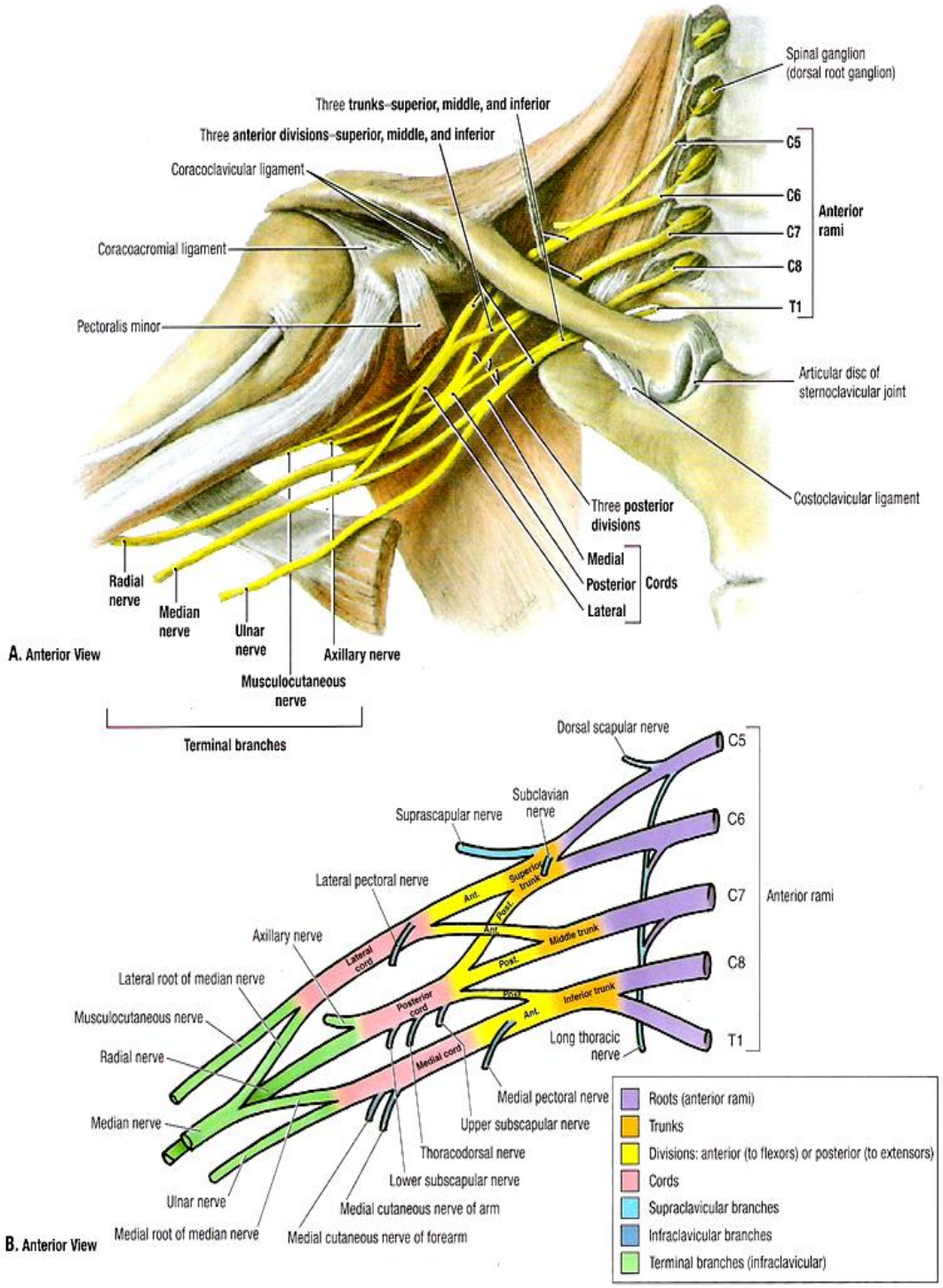
Brachial Plexus is one of the most commonly used peripheral nerve blocks in clinical practice. So knowledge of the formation of the brachial plexus and of its distribution is absolutely essential for the effective use of brachial plexus block for surgeries of the upper limb. Absolute familiarity with the Vascular, Muscular and fascial relationship of the brachial plexus throughout its formation and distribution is equally essential for the mastery of various techniques of brachial plexus anaesthesia.

In its course from the intervertebral foramina to the upper arm, the fibres that constitute the plexus are composed consecutively of roots, trunks, cords, divisions and terminal nerves which are formed through a complex process of combining, dividing, recombining and finally redividing.

FORMATION OF THE PLEXUS:

ROOTS:

The plexus is formed by the anterior primary rami of 5th to 8th cervical plexus together with the bulk of the 1st thoracic nerve (C8-T1).



6.23 Brachial plexus

A. Dissection. B. Schematic illustration.

In addition, there is frequently a combination from C4 to the 5th cervical roots and another below the T2 to the 1st thoracic nerve. Occasionally the plexus is mainly derived from C4-C8 (prefixed plexus) or from C6-T2 (post fixed plexus).

TRUNKS:

The five roots of the brachial plexus emerge from the intervertebral foramina. They lie in the gutter between the anterior and posterior tubercles of the corresponding transverse process. All five roots then become sandwiched between Scalenus anterior and Scalenus medius. Here the roots of C5 and C6 unite into the upper trunk, the root of C7 continues as the middle trunk and those of C8 and T1 into the lower trunk. Each trunk divides behind the clavicle, into anterior and posterior divisions which unite in the axilla to form cords.

CORDS:

The six divisions stream into axilla and there join up into three cords; lateral, medial and posterior. These cords are composed as follows:

The union of the anterior divisions of the Upper and middle trunks form the lateral cord. The medial cord represents the continuation of the anterior division of the lower trunk. The posterior cord comprises of the posterior divisions of all the three trunks.

The composition of brachial plexus can be summarised as follows:

1. Five roots (between the scalene muscles) - the anterior primary rami of C5-C8 and T1.

2. Three trunks (in the posterior triangle)
    a) Upper trunk C5 and C6
    b) Middle trunk C7 alone
    c) Lower trunk C8 and T1

3. Six divisions (behind the Clavicle) Each trunk divides into an anterior and posterior division.

4. Three cords (within the axilla)
    a. Lateral Cord - the fused anterior divisions of the upper and middle trunks C5-C7

b. Medial Cord - the anterior division of the lower trunk C8-T1

c. Posterior Cord formed by the union of the posterior divisions of all three trunks C5-T1

RELATIONS OF THE BRACHIAL PLEXUS:

ROOTS:

Lie between the Scalenus anterior and Scalenus medius. The roots of the Plexus lie above the second part of the subclavian artery.

TRUNKS:

In the Posterior triangle, the trunks of the plexus invested in a sheath of prevertebral fascia, are superficially placed, being covered by skin, platysma and deepfascia.

The upper and middle trunks lie above the subclavian artery as they stream across the first rib, but the lower trunk lie behind the artery and may groove the rib immediately posterior to the subclavian groove.

DIVISIONS:

At the lateral border of the first rib, the trunks bifurcate into divisions which are situated behind the clavicle, subclavius muscle and the suprascapular vessels.

CORDS:

The cords are formed at the apex of the axilla and become grouped around the axillary artery.

THE INTERSCALENE SHEATH:

As the roots emerge in the groove between the transverse process of the tubercle, they lie in a fibro fatty space between two layers of fibrinous sheath.

Posterior Sheath from the posterior tubercles cover the front of the medius, anterior sheath from anterior tubercles cover the posterior aspect of the Scalenus anterior. The sheath extends into the axilla around the plexus.

Significance of this space is that the local anaesthetic can be injected to produce block at various sites by interscalene, subclavian perivascular or the axillary approach.

SYMPATHETIC SUPPLY:

Close to the emergence the 5th and 6th Cervical nerves receive a grey ramus from the middle cervical sympathetic ganglion. The 7th and

8th cervical nerves each receive a grey ramus from the inferior cervical ganglion and from T1ganglion

BRANCHES:

Branches are given off from roots, trunks and cords.

1. Branches from the roots:
    a) Nerve to the serratus anterior C5, C6 and C7
    b) Muscular branches to
        - Longus cervices C5 - C8
        - Three Scalene C5 - C8
        - Rhomboids C5
    c) Twig to the Phrenic nerve C5
2. Branches from the trunks:
    a) Suprascapular nerve C5-C6
    b) Nerve to subclavius C5-C6
3. Branches from the Cords:
    a) Lateral Cord
        - Lateral Pectoral nerve C5-C7
        - Lateral head of median nerve C5-C7
        - Musculocutaneous nerve C5-C7

b) Medial Cord

- Medial Pectoral nerve C8 - T1
- Medial head of median nerve C8 - T1
- Medial Cutaneous nerve of arm C8 - T1
- Medial Cutaneous nerve of forearm C8 - T1
- Ulnar nerve of arm C7, C8 - T1

c) Posterior Cord

- Upper Subscapular nerve C5-C6
- Lower Subscapular nerve C5-C6
- Nerve to latissimus dorsi C6, C7, C8
- Axillary nerve C5-C6
- Radial nerve C5, C6, C7, C8, T1

ANATOMIC CONSIDERATIONS OF THE INTERSCALENE SPACE:

The roots of the brachial plexus, after leaving the transverse process of the corresponding cervical vertebrae, descend in between the scalenus anterior and medius in the posterior triangle of the neck.

Scalenus anterior arises from the anterior tubercles of the transverse processes of C3- C6 Vertebrae. It is inserted into the scalene

tubercle on the inner border of the first rib. The muscle lies anterior to the plexus and at its insertion lies anterior to the subclavian artery which separates the plexus from its insertion. Scalenus medius arises from the posterior tubercles of the six lowest cervical vertebrae and is inserted into the upper surface of the first rib behind the groove made by the brachial plexus and the subclavian artery. Thus the plexus lies in front of the muscle.

The first rib lies in an almost horizontal plane being inclined slightly downwards and forwards. It passes below the clavicle at about the junction of its inner and middle thirds. The upper surface of first rib has two transverse grooves - an anterior one for the subclavian vein and a posterior one for the subclavian artery and the lowest trunk of the brachial plexus. On the inner border between the grooves is the scalene tubercle.

Brachial line runs in a straight line from the transverse process of the C6 vertebra to the axillary artery in the axilla. It runs inferolaterally at an angle of 45degree from the horizontal plane and slightly forwards at 15 degree.

Techniques of Brachial Plexus Block:

Surgical anaesthesia of the upper extremity and shoulder can be achieved following neural blockade of the brachial plexus at various sites. The various approaches that can be used for this blockade is as follows:

i) Interscalene approach

ii) Supraclavicular approach

   a) Classic approach

   b) Plumb bob technique

   c) Supraclavicular Perivascular technique

iii) Axillary approach

iv) Infraclavicular approach

1. INTERSCALENE BRACHIAL PLEXUS BLOCK:

In this technique plexus is blocked at the level of the C6 vertebra. By standing at the side of the patient and after locating the interscalene groove, an intradermal wheal is raised at the point of needle insertion which is at the level of the cricoid cartilage. A 22gauge 3.5 cm short bevel needle is inserted “at right angles to the skin in all planes” i.e. dorsal to the horizontal planes. The needle is advanced slowly until

paraesthesia sought in the shoulder or a nerve stimulator is used to evoke contractions in the deltoid or biceps brachialis muscle.20 -40 ml of local anaesthetic injected after repeated aspiration to detect inadvertent entry into vertebral artery or dural cuff.

COMPLICATIONS:

1. Subarachnoid injection
2. Epidural blockade
3. Intravascular Injection
4. Pneumothorax
5. Phrenic nerve block

2. SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK:

A. CLASSICAL SUPRACLAVICULAR BLOCK OF KULENKAMPFF:

In the classic approach, the needle insertion site is 1 cm superior to the clavicular mid point. The needle is inserted in a plane parallel to the patient neck and head. The needle will contact the rib at a depth of 3 to 4 cm. The needle is walked over the rib until paraesthesia is elicited. After careful aspiration the local anaesthetic is injected.

B. PLUMB BOB SUPRACLAVICULAR BLOCK:

The brachial plexus at the level of the first rib lies posterior and cephalic to the subclavian artery. Once this skin mark has been placed immediately superior to the clavicle at the lateral border of the sternomastoid muscle as it is inserted into the clavicle, the needle is inserted at a 90 degree angle to the table top.

The local anaesthetic is injected after eliciting paraestehsia. The name Plumb bob was chosen for this technique because if one suspends a Plumb bob over the entry site, needle inserted through that point will result in contact with the brachial plexus in most patients.

C. SUBCLAVIAN PERIVASCULAR TECHNIQUE OF WINNIE AND COLLINS:

The interscalene groove is palpated at its most inferior point, which is just posterior to the subclavian artery pulse. The needle is directed just above and posterior to the subclavian pulse and directed caudally at a flat angle against the skin. The needle is advanced until paraesthesia is elicited and the local anaesthetic is injected after careful aspiration.

COMPLICATIONS:

1. Pneumothorax
2. Horner’s syndrome
3. Phrenic nerve block
4. Haemothorax and Haematoma formation.

3. INFRACLAVICULAR TECHNIQUE:

This is the preferred technique for the surgeries of elbow and lower arm because spread of local anaesthetic is kept below the clavicle. This technique blocks the brachial plexus at the level of cords. The needle is inserted 1 inch beneath the midpoint of the clavicle. It is then directed laterally from this site at a 45 degree angle away from the chest wall and towards the humeral head or the coracoid process. Once paraethesia is elicited, the local anaesthetic is injected.

COMPLICATIONS:

1. Pneumothorax
2. Haemothorax
3. Chylothorax with a left side block

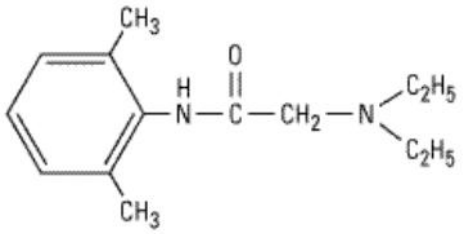
4. AXILLARY BRACHIAL PLEXUS BLOCK:

The pulsations of the axillary artery are best felt high in the axilla between the coracobrachialis and pectoralis major muscle. The needle is inserted just superior to the artery until the resistance of the fascial sheath is felt and a pop indicated the correct needle placement.

COMPLICATIONS:

1. Intra arterial Injection.
2. Post Operative neuropathy
3. Hematoma and Infection.

PHARMACOLOGY OF LIGNOCAINE



Lidocaine

Lignocaine was synthesized in 1943 in Swedan by Lofgren and was introduced into clinical practice in 1948.

DESCRIPTION

Lignocaine hydrochloride is 2-diethylamino-aceto-2'6xylilide hydrochloride monohydrate. It appears as a white crystalline powder which is odourless. It is very soluble in water, freely soluble in chloroform and in ethanol and is practically insoluble in ether.

Molecular formula - C14H22 N20 Hcl.H20

Molecular weight - 288.8

Lignocaine hydrochloride injection is a sterile, isotonic solution containing lignocaine hydrochloride B.P., 1% or 2%, and sodium chloride, B.P., in water for injection. Lignocaine is a weak base with amphiphillic property. A hydrophilic amine on one side and a lipophillic

lipophillic form (B+), reionises in the axoplasm and approaches the LA receptor through the intracellular mouth of the channel. It is the cationic form of local anaesthetic (BH+), which primarily binds to the LA receptor. The receptor has higher affinity or is more accessible to local anaesthetic in the activated state compared to the resting state. Binding of LA to its receptor stabilizes the channel in the inactive state and thus reduces the probability of channel opening.

Action of receptors within the sodium channel accounts for 90% of nerve blocking effect. Nonspecific membrane expansion accounts for the remaining 10% of the action and is analogous to the electrical stabilization produced by a number of non-polar, purely lipid solvable substances such as barbiturates, general anaesthetics and benzocaine.

PHARMACOLOGICAL ACTION:

1. **LOCAL** - Minimal local irritant action and blocks sensory nerve endings, nerve trunks, neuro-muscular junction, ganglionic receptors.
2. **REGIONAL** - Autonomic fibers are generally more susceptible than somatic fibers. Among the

somatic afferents, the order of blockade is pain, temperature, touch ,deep pressure.

3. **SYSTEMIC** - Effect is mainly on CVS or CNS.

CVS : In cardiac tissue, a therapeutic serum concentration (1.5 to 6. micrograms / ml) of lignocaine will produce the following effects:

a. Depression of slow spontaneous depolarization (phase 4), that is the automaticity of isolated, non-polarised purkinjee fibres, while having little effect on conduction velocity, membrane responsiveness or cardiac output. Automaticity induced by stretch, hypoxia or catecholamines can also be suppressed by lignocaine.

b. Shortening of action potential period and effective refractory period of purkinjee and ventricular cells.

Thus it has a stabilizing effect on cell membrane of cardiac tissue. It also stabilizes aberrant conduction.

CNS : Low plasma concentration of LA are likely to produce numbness of tongue and circumoral tisses. As plasma concentration increases it crosses blood-brain-barrier and produces restlessness, vertigo, tinitus and difficulty in focusing. Then slurred speech and

skeletal muscle twitching occur. Lignocaine causes drowsiness before seizures. Seizures are classically followed by CNS depression, which may be accompanied by hypotension and apnoea.

PHARMACOKINETICS

Following IV injection, the blood level of lignocaine declines with a half-life of 7 to 10 mins, within the first hour due to rapid distribution into various tissues including the heart. After this initial phase, the half-life is 90 to 120 mins (metabolism and excretion). Absorption is slow in regional anaesthesia.

METABOLISM AND EXCRETION

The principle metabolic pathway of lignocaine is oxidative dealkylation in the liver to mono ethyl glycinexylidine following by hydrolysis of this metabolite to xylidine. Mono ethyl glycinexylidine has approximately 80% of the activity of lignocaine for protecting against cardiac dysrhythmias. This metabolite has a prolonged elimination half time. Xylidine has approximately 10% of the activity of lignocaine.

Hepatic disease or decrease in hepatic flow, which may occur during general anaesthesia, decreases the rate of metabolism of lignocaine. Excretion is through the kidneys. Approximately 90% of the dose is excreted as metabolites and less than 10% is excreted unchanged in the urine.

DOSAGE

For regional anesthesia: 3mg/kg, with adrenaline 7mg/kg. For cardiac arrythmias, therapeutic serum concentration of lignocaine is 5 to 20 micromol/L or 1.5 to 6.0 micrograms/ ml. A single intravenous dose of 1mg/kg should be given over 1 to 2 mins, to obtain therapeutic blood levels rapidly. The initial effect will occur in 2 to 4 mins, and may last as long as 20 mins. This should be followed within 10 mins, by a continuous infusion at the rate of 2 to 4 mgs/min. The initial dose may be repeated by two more injections at 15 to 20 min intervals to maintain therapeutic blood levels but no more than 300mg of lignocaine should be administered within a 1 hr period. Since it has a very narrow therapeutic window, infusion should promptly stopped when there is an undue prolongation of PR interval or QRS complex.

To attenuate the cardiovascular stress response to intubation, lignocaine 1.5mg/kg IV 3 min prior to laryngoscope should be given.

ADVERSE EFFECTS /TOXICITY:

Due to high plasma levels as a result of excessive dosage, rapid absorption, delayed elimination or inadvertent IM injection during local anaesthetic use.

1. CNS: Lightheadedness, disorientation, confusion, psychois, nervousness, agitation, drowsiness, euphoria, tinnitus, blurred vision, slurred speech, numbness, twitching, tremors, convulsions, unconsiousness, seizures, coma, respiratory depression and arrest.

2. CVS: Hypotension, CVS collapse, arrhythmias, heart block and bradycardia which may lead to cardiac arrest. Meth-hemoglobineamia may occur following IV administration.

3. HYPERSENSITIVITY: Rare with lignocaine.

4. NEUROLOGICAL SYSTEM : Persistent anaestheisa, paresthesia, weakness, paraplegia of lower extremeities and loss of sphincter control may occur.

PRECAUTIONS

The safety and effectiveness of lignocaine depends upon proper dosage, correct technique, adequate precautions and readiness for emergencies.

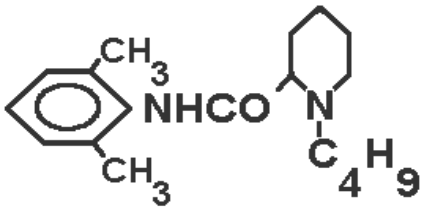
1. Lignocaine should be given cautiously in patients with severe bradycardia, cardiac conduction disturbances, severe digitalis intoxication, severe shock and hypovolemia.
2. Serum Potassium level should be normalized prior to administration of lignocaine as antiarrhythmic drugs may be ineffective in hypokalemic patients.

DRUG INTERACTIONS

Propanolol and metaprolol reduce the metabolism of intravenously administered lignocaine. It is possible that this effect will be repeated with other beta - adrenergic blockers.

1. Phenytoin, phenobarbitone, primidone and carbamazapine appears to enhance the metabolism of lignocaine, possible due to an induction of microsomal enzyme.
2. Lignocaine prolongs the duration of suxamethonium.

PHARMACOLOGY OF BUPIVACAINE



Bupivacaine is an amide linked local anaesthetic. It is a hydrochloride salt of 1-butyl-N-(2, 6-dimethylphenyl) piperidine-2-carboxamide and is presented as a racemic mixture.

It was synthesized by Ekenstam in 1957.First report of its use was published in 1963 by Telivuo. It is derived from Mepivacaine and is very stable compound and may be autoclaved repeatedly.

Pka	-	8.1
Molecular weight	-	288
Protein binding	-	95%
Lipid solubility	-	28
Elimination half life	-	210 minutes
Toxic plasma concentration	-	>1.5µg/ml
Approximate duration of action	-	175minutes

Availability:

Ampoules

- 0.5% Bupivacaine hydrochloride with dextrose(Heavy) 4cc
- 0.5% Bupivacaine hydrochloride (plain)

Vials

- 0.25% and 0.5% Bupivacaine hydrochloride 20cc

Dosage

- Maximum dosage 3mg/kg body weight.

Uses:

- Spinal anaesthesia
- Epidural anaesthesia
- Caudal anaesthesia
- Continuous epidural anaesthesia
- Peripheral nerve block

Onset time and duration of action

Site of action	**Onset (minutes)**	**Duration (minutes)**
Intrathecal	5	90-120
Epidural	15-20	165-225
Brachial plexus	10-20	600

Pharmacokinetics:

Once injected intrathecaly, it gets absorbed by the nerve rootlets and results in the desired effect. It is rapidly absorbed from the site of injection, but the rate of absorption depends on the vascularity at the site and presence of vasoconstrictors.

High lipid solubility of bupivacaine makes it easy for nerve and vascular tissue penetration. 80-95% of the absorbed bupivacaine binds to the plasma proteins.

Distribution:

1. Rapid distribution phase: (α)

   In this phase the drug is distributed to highly vascular region, t½ of α - being 2. 7 minutes.

2. Slow disappearance phase: (β)

   In this phase the drug is distributed to slowly equilibrating tissues, t1/2 of β being 28minutes.

3. Biotransformation and excretion phase: (δ)

   T1/2 of δ is 3.5hours. Clearance is 0.47 litre/minute.

Biotransformation:

Possible pathways of metabolism of bupivacaine include earomatic hydroxylation and conjugation. Only the N-dealkylated metabolite, N-desbutyl bupivacaine has been measured in blood (or) urine after epidural (or) spinal anaesthesia. Alpha-1 acid glycoprotein is the most important plasma protein binding site of bupivacaine and its concentration is increased by many clinical situations including post operative trauma.

Excretion:

It is through the kidney, 4-10% of the drug is excreted unchanged.

Mode of Action:

a) Site of action:

i) Peripheral nerve rootlet , fine nerve filaments

ii) The spinal nerve rootlet fine nerve filaments having a large surface area are exposed to the local anaesthetics

iii) Posterior and lateral aspects of the spinal cord itself.

b) Sodium Channel blockade:

They impede sodium ion access to the axon interior by occluding the transmembrane sodium channels thus delaying the process of

depolarization and axon remains polarized. It is a nondepolarization blockade.

Pharmacodynamics:

It has got a longer duration of action but a slower onset.

Cardio vascular system:

It depresses myocardial automaticity (Spontaneous Phase IV depolarization) and reduce the duration of the refractory period. Myocardial contractility and conduction velocity are also depressed at high concentrations. It causes some degree of arteriolar vasodilatation. The ensuing combination of bradycardia, heart block, and hypotension may culminate in cardiac arrest.

Respiratory System:

It relaxes bronchial smooth muscle. Apnea can results from phrenic and intercostal nerve paralysis or depression of the medullary respiratory center following direct exposure to drug.

Toxicity:

Toxicity is related to plasma level of unbound drug and more likely due to an inadvertent intravenous injection. Systemic toxicity reactions primarily involve central nervous system and cardio vascular

system. The blood level required to produce central nervous system toxicity is less than that required to produce circulatory collapse.

Central Nervous System Toxicity:

Early symptoms are circumoral numbness, tongue paresthesia, and dizziness. Sensory complaints include tinnitus and blurred vision. Excitatory signs (eg, restlessness, agitation, nervousness, paranoia) often precede central nervous system depression (eg, slurred speech, drowsiness, unconsciousness). Muscle twitching heralds the onset of tonic clonic seizures.

Respiratory arrest often follows. The excitatory reactions are a result of selective blockade of inhibitory pathways.

Cardiovascular System Toxicity:

The rate of depolarization in fast conducting tissue of purkinje fibres and ventricular muscle is decreased. The rate of recovery of bupivacaine induced block is slower than that of lignocaine.

Extremely high concentration of the drug causes sinus bradycardia, hypotension, atrioventricular heart block, idioventricular rhythms, and life threatening arrhythmias such as ventricular tachycardia, ventricular fibrillation and cardiac arrest.

aromatic residue on the other side and are joined through an amide linkage.

MECHANISM OF ACTION:

Local anaesthetics block the nerve conduction by decreasing the entry of sodium ions during upstroke of action potential. As the concentration of local anaesthetic is increased the rate of rise of action potential and maximum depolarization decreases causing slowing of conduction. Finally local depolarization fails to reach the threshold potential and conduction block ensues. The local anesthetics interact with a receptor situated within the voltage sensitive sodium channel and raise the threshold of channel opening.

Sodium channel has an activation gate(A) near its extracellular mouth and an inactivation gate(I) at the intracellular mouth. In the resting state, the activation gate is closed. Threshold depolarization of the membrane opens the activation gate allowing sodium ions to flow in along the concentration gradient. Within a few milliseconds inactivation gate closes and ion flow ceases.

The local anaesthetic recepter is located within the channel in its intracellular half. Local anaesthetic traverses the membrane in its

PHARMACOLOGY OF DEXAMETHASONE

Dexamethasone is a very potent and highly selective glucocorticoid. It is a long acting drug.

PHARMACOLOGICAL ACTIONS

1. Carbohydrate and protein metabolism

Glucocorticoids promote glycogen deposition in the liver by inducing hepatic glycogen synthetase and promoting glyconeogenesis. It inhibits glucose utilisation in peripheral tissues. This along with increased glucose release causes insulin resistance and a diabetes like state. It causes protein breakdown and amino acid mobilization from peripheral tissues, responsible for side effects like muscle wasting. The amino acids are funneled into the liver and used for glyconeogensis. Excess urea is produced and these cause negative nitrogen balance.

Fat metabolism

This action is primary permissive in nature. It promotes lipolysis due to glucagon, growth hormone, adrenaline and thyroxine. Cyclic adenosine mono phosphate (CAMP) induced breakdown of triglycerides are enhanced.

Fat areas in various sites respond differently and redistribution of fat occurs. More fat is deposited in the face, neck, shoulder – (moon face, fish mouth and buffalo hump), and there is loss of subcutaneous fat over the extremities. The explanation to this is because the peripheral adipocytes are less sensitive to insulin, and corticosteroid enhanced lipolytic action of adrenaline and growth hormone predominates.

Calcium metabolism

It inhibits the intestinal absorption of calcium and increase renal excretion of calcium. There is also loss of calcium from bone indirectly due to loss of osteoid in chronic use. Spongy bones (vertebra, ribs) are more sensitive.

Water excretion

Effect on water excretion is independent of action on sodium transport, in maintaining GFR. In adrenal insufficiency the capacity to excrete a water load is considerably reduced. Glucocorticoid enhances the secretory capacity of renal tubules.

Cardiovascular system

Glucocorticoids restrict capillary permeability and maintain tone of arteries and myocardial contractility. It has permissive effect on

pressor action of adrenaline and angiotensin. They also play a role in development of hypertension and should be cautiously used in hypertensives.

Skeletal muscles

Optimal level of corticosteroids are needed for normal muscle function. Weakness may occur in both hypo and hypercortism but the effects may be different.

Hypocortism– weakness due to hypodynamic circulation

Hypercortism– excess glucocorticoid action → muscle wasting → myopathy→ weakness.

Central nervous system

Mild euphoria is common with pharmacological doses of glucocorticoids. This is due to a direct effect on brain, independent of relief of disease symptoms, sometimes progresses to cause increased motor activity, insomnia, anxiety or depression. It also maintains the level of sensory perception and normal level of excitability of neurons. High doses, lower seizure threshold in epileptics.

Stomach

Secretion of gastric acid and pepsin is increased and may aggravate peptic ulcer.

Lymphoid tissue and blood cells

Glucocorticoid drive can raise the rate of destruction of lymphoid cells (T cells are more sensitive than B cells). However a marked lytic response is shown by malignant lymphatic cells – usually in lymphomas. Corticosteroid increases the number of RBCs, platelets and neutrophils in the circulation. They decrease lymphocytes, basophils and eosinophils. These are due to sequestration of cells. The count becomes normal in 24 hrs.

Inflammatory responses

Irrespective of type of injury or insult, the attending inflammatory response is suppressed by glucocorticords. This is the basis of most of their clinical uses. It lowers all stages of inflammation. The action are direct and even local application is possible.

The cardinal signs of inflammation such as redness, heat, swelling and pain are suppressed. Corticosteroids are only palliative, the

underlying disease processes continues while manifestations are dampened.

Immunological and allergic response

They cause greater suppression of cell mediated immunity in which T cells are primarily involved example: delayed hypersensitivity and graft rejection. It decreases the release of interleukin-1 and interleukin -2.

Pharmacokinetics

Dexamethasone is a water soluble ester, in the form of dexamethasone sodium phosphate. It has an oral, intramuscular or intravenous preparation. It acts rapidly and attain high concentration in tissue fluids. Dexamethasone is mainly metabolized in the liver by hepatic microsomal enzymes. The t 1/2 of dexamethasone in greater than 36 hrs, its action starts within 30 minute of injection and action persists even after the drug disappears from the circulation.

Drug interaction

Phenobarbitone and phenytoin induce metabolism of dexamethasone to decrease its therapeutic effect

USES

1. **Arthritis:** Used in rheumatoid arthritis in conjunction with NSAIDs, arthritis in rheumatic fever and in gouty arthritis.

2. **Collagen diseases:** In disease like systemic lupus erythematosus, polyarteritis nodosa, dermatomyositis, this drug may be life saving.

3. **Severe allergic reactions:** Used in anaphylaxis for short periods, in angioneurotic edema, urticaria, serum sickness.

4. **Autoimmune diseases:** Autoimmune hemolytic anemia, thrombocytopenia, active chronic hepatitis respond to corticosteroids.

5. **Bronchial asthma:** Used intravenously in status asthmaticus.

6. **As an adjunct to drug in nausea and vomiting:** As it prevent the release of inflammatory mediators and prostoglandins, they modulate neuronal activity and have a membrane stabilising action. It has a minimal action in nausea and vomiting. It increases the efficacy of the drug which is co-administered.

7. **Eye disease:** Allergic conjuctivitis, iridocyclitis, keratitis

8. **Intestinal disease:** In diseases like crohns, ulcerative colitis, celiac disease with remissions and exacerbations.

9. **Cerebral edema:** In cerebral edema due to meningitis and tumours. Dexamethasone is preferred because, it does not have sodium retaining capacity. Large doses given in spinal injury can reduce neurological sequalae.

10. **Malignancies:** As a component of chemotherapy, in ALL, Hodgkin's and other lymphomas.

11. **In organ transplantations and skin allograft:** To prevent rejection.

12. **To test adrenal** –Pituitary axis function.

Contraindications for chronic use

These are only relative contraindications since, the steroid dexamethasone is a life saving drug, they are, peptic ulcer, diabetes mellitus, hypertension, viral and fungal infections, tuberculosis and other infections, osteoporosis, herpes simplex keratitis, psychosis, epilepsy, congestive heart failure, renal failure.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

1. Yadav RK, SahBP, KumarP, SinghSN[46], compared effectiveness of addition of dexamethasone vs neostigmine to lignocaine ,adrenaline mixture for brachial plexus block in providing perioperative analgesia. 90 patients were randomized in 3 groups . group A (lignocaine 1.5% with adrenaline 24 ml) group B (lignocaine 1.5% with adrenaline + 500mic gm neostigmine) and group C (lignocaine 1.5% with adrenaline +4mg dexamethasone). Mean onset of analgesia 4.6 min,4.4min,3.8 min in group A,B and C respectively and the mean onset of motor blockade were 7.7 min,7min,6min in group A,B, and C respectively. similarly mean complete sensory block were 10.6mins, 10.4 mins, 8.9mins and mean complete motor blockade were 17.3mins, 17.2mins and 14.7mins respectively in group A, group B, and group C. Duration of analgesia was mean 176.5min, 225.7min and 454.2mins in group A, group B and group C respectively. They concluded that onset of sensory and motor block is faster and the duration of analgesia is longer in dexamethasone group and also need less number of rescue analgesic requirement.

2. Shrestha BR, Maharjan SK, Tabedar S[34], compared the analgesic efficacy of local anesthetic with and without dexamethasone

in supraclavicular brachial plexus block. 40 patients undergoing upper limb surgeries were randomly selected and divided into 2 groups of 20 each. Group A received mixture of 2% lidocaine with 1:200000 adrenaline and bupivacaine 0.5% for a total volume of 40 to 50 ml. group B received local anesthetic mixture with dexamethasone 4 to 8 mg. The onset of action and duration of analgesia in the two groups were compared and any complication of the procedure were noted. Onset of action was 18.15 min in group A and 14.5 min in group B. duration of action in group A was mean 3.16 hrs ,in group B mean 12.75 hrs They concluded that the onset of action was faster and duration of analgesia is prolonged in the dexamethasone group than the other group and there were no complications.

3. **Ali movafegh, Mehran razazian, Alipasha meysamie**[27] studied the analgesic efficacy of dexamethasone with lidocaine in axillary brachial plexus blockade. 60 patients scheduled for upper limb hand and fore arm surgeries under axillary brachial plexus block were randomly allocated to receive either 34 ml lidocaine 1.5% with 2ml of normal saline .(30 patients group A) or 34 ml lidocaine 1.5% with 2ml of dexamethasone.(30 patients group B) . The duration of sensory blockade in group A is mean 98 min and in group B 242 min. The duration of

motor blockade in group A is mean 130min and group B is mean 310min. They concluded that the addition of dexamethasone to lidocaine 1.5%solution in axillary brachial plexus block prolongs the duration of sensory and motor blockade.

4. Simon J. Parrington, Dermot O' Donnell, Vincent W.S. Chan, Rajiv subramanyam and Richard brull[36] analysed the efficacy of dexamethasone added to mepivacaine in supraclavicular brachial plexus block in providing post operative analgesia.45 adult patients undergoing elective upper limb surgeries were randomized to receive either 30ml mepivacaine 1.5% plus dexamethasone or 30ml mepivacaine 1.5% plus 2ml normal saline. Mean duration of analgesia was significantly prolonged in dexa group (332mins) than normal saline group (228mins). The onset of action of sensory and motor block was similar in both groups. Complications were minor and transient and did not differ between both groups at 2weeks post operatively. They concluded that addition of 8mg dexamethasone to 1.5% 30 ml mepivacaine prolongs the duration of analgesia, but does not reduce the onset of sensory and motor blockade after ultrasound guided brachial plexus block compared with mepivacaine alone.

5. Shrestha BR, Maharjan SK, Shrestha S, Gautam B, Thapa C, Thapa PB, Joshi MR[35] compared the efficacy of tramadol and dexamethasone as an adjuvant to bupivacaine in supraclavicular brachial plexus block. 60 patients were randomly divided into 2groups, group A received 0.5% bupivacaine 2ml/kg with tramadol 2mg/kg and group B received 0.5% bupivacaine with dexamethasone 8mg.Mean onset of motor blockade in group A is 12.90mins and in group B is 13.43mins.The mean onset of sensory blockade is 18.47mins in group A and 16.76mins in group B. The duration of post operative analgesia was recorded in both groups using pain VAS score which was determined by maximum VAS score of 8 to 10 and when patient demands additional analgesics. The mean duration of post operative analgesia in dexamethasone group was 1028 minutes while in tramadol group it was 453.17 minutes. They concluded that dexamethasone with local anesthetic shortens the onset of analgesia and prolongs postoperative analgesia significantly than tramadol.

6. Franco CD, Vieira ZE (2000) did a study on Subclavian Perivascular Block and its success using a nerve stimulator. They concluded that the Subclavian perivascular technique consistently provides an effective block for surgery on the upper extremity. At the

site of injection the plexus is reduced to its smallest components and the sheath is reduced to its smallest volume which explains in greater part the success obtained with this block.

7. Brown DL (1993) did a study on brachial plexus anaesthesia and analysed the various sites at which the plexus can be blocked. They studied the supraclavicular, interscalene, infraclavicular and axillary approaches. They concluded that the supraclavicular block produces anaesthesia of the entire upper extremity in the most consistent and efficient manner than any other brachial plexus block technique.

8. Lanz.E, Theiss D, Jankovic D (1983) studied the extent of blockade using various techniques of brachial plexus blocks. The extent of sensory and motor blockade was assessed using 0.5% bupivacaine. They concluded that the subclavian Perivascular approach of Winnie resulted in a homogenous blockade of the nerves of the brachial plexus.

9. Winnie and Ramamoorthy (1977)[(44)] postulated that the trunk of brachial plexus are arranged so that the central fibres are longest supplying the extremities of the limb, while the shorter fibres are arranged more peripherally as their area of supply is more proximal. Winnie groups the fibres into two: the Peripheral Mantle bundle which

contains the outer motor and inner sensory fibres corresponding to all the early branches of the brachial plexus being motor: and a central core bundle with the outer motor fibres supplying muscles of the forearm and the inner sensory fibres carry sensation from the hand.

Thus the order of blockade is as follows:

Loss of motor power to the shoulder and upper arm; loss of sensation in the upperarm; loss of motor power of the forearm; and loss of sensation of the hand.

10. Lanz.E, Theiss (1979) compared the supraclavicular and the interscalene approach of brachial plexus block. They concluded that with the Supraclavicular block, motor as well as sensory blockade of all the nerves of the brachial plexus occurred with about the same frequency. Following both the techniques, blockade developed from the proximal to distal with motor blockade preceding the sensory block.

11. Viera PA, Pulai I , Tsao GC, Manikantan P, Keller B, Connelly B[(42)] analysed the efficacy of dexamethasone with bupivacaine in ultrasound guided interscalene brachial plexus block for providing perioperative analgesia. This prospective, randomized, double-blind investigation was performed on 88 individuals undergoing shoulder

arthroscopy. Patients received interscalene brachial plexus block using 20 ml of 0.5%bupivacaine with 1: 200 000 epinephrine and clonidine 75 μg. Patients were randomly assigned to receive either dexamethasone 8 mg or 0.9% NaCl as an adjuvant to the mixture. After discharge, patients recorded pain scores and analgesic consumption in a diary and estimated the time at which they perceived that the sensory block from the interscalene brachial plexus block resolved. In their study dexamethasone prolonged median sensory (1457 vs. 833 min, $P<0.0001$) and motor (1374 vs. 827 min, $P < 0.0001$) blockade compared with the control. At 24 h, the dexamethasone group had lower median verbal analogue scale scores compared with control (3.0 vs. 6.0). They noted that the addition of dexamethasone with bupivacaine increases the duration of analgesia in ultrasound guided interscalene brachial plexus blockade.

12. K. C. Cummings , D. E. Napier kowski, I. Parra-Sanchez, A. Kurz, J. E. Dalton, **J. J. Brems and D. I. Sessler**[20] Effect of dexamethasone on the duration of interscalene nerve blocks with ropivacaine or bupivacaine**.** In a double-blinded trial utilizing single-injection interscalene block, patients were randomized to one of four groups: (i) ropivacaine: 0.5% ropivacaine; (ii) bupivacaine: 0.5%

bupivacaine; (iii) ropivacaine and steroid: 0.5% ropivacaine mixed with dexamethasone 8 mg; and (iv) bupivacaine and steroid: 0.5% bupivacaine mixed with dexamethasone 8 mg. The primary outcome was time to first analgesic request after post-anaesthesia care unit discharge. They noticed dexamethasone significantly prolonged the duration of analgesia of both ropivacaine[11.8*vs* 22.2 h, $P<0.001$] and bupivacaine [14.8 vs 22.4$P<0.001$] in their study. They concluded that the addition of dexamethasone 8mg to ropivacaine or bupivacaine prolongs the duration of analgesia.

13. Youn-Jin Kim, Jong-In Han, Hee-Jung Baik, Guie-Yong Lee[47] The purpose of their study is to evaluate the effect of the addition of 5mg dexamethasone to 10 ml of 5 mg/ml levobupivacaine on postoperative analgesic effects of ultrasound guided interscalene brachial plexus block for arthroscopic shoulder surgery under general anesthesia. In 45 patients scheduled for arthroscopic shoulder surgery underwent general anesthesia, interscalene brachial plexus block was preoperatively performed with 10 ml of 5mg/ml levobupivacaine under the guidance of ultrasound and nerve stimulator. Patients were randomly allocated to receive same volume of normal saline (Group I), 5 mg of dexamethasone (Group II), or 1: 400,000 epinephrine (Group III) as an

adjuvant to the mixture. A blind observer recorded verbal numerical rating scale (VNRS) at 0, 1, 6, 12, 24, 48h after operation, total analgesic consumption, sleep quality, complication, and patients` satisfaction. All patients had successful interscalene brachial plexus blocks and excellent analgesic effects less than 4 of VNRS up to discharge time. VNRS in Group II at 12h and 48h was statistically much lower than in Group I and III. There were no differences in total analgesic consumption, sleep quality, complications, and patients` satisfaction. They conclude that the addition of 5mg of dexamethasone to 10 ml of 5 mg/ml levobupivacaine in intersclane brachial plexus block showed improvement of postoperative analgesia for arthroscopic shoulder operation without specific complication.

14. Islam SM, Hossain MHMD, Maruf AA[17] evaluated the effect of dexamethasone added to local anaesthetics on the onset and duration of supraclavicular brachial plexus block. Sixty adult patients undergoing various orthopaedic surgeries on forearm and around the elbow under supraclavicular brachial plexus block were selected and divided into 2 groups of 30 each. In group-A patients received 35 ml of mixture of lignocaine 2%, bupivacaine 0.5% while in group-B patients received the same amount of local anaesthetics with dexamethasone (8 mg). The

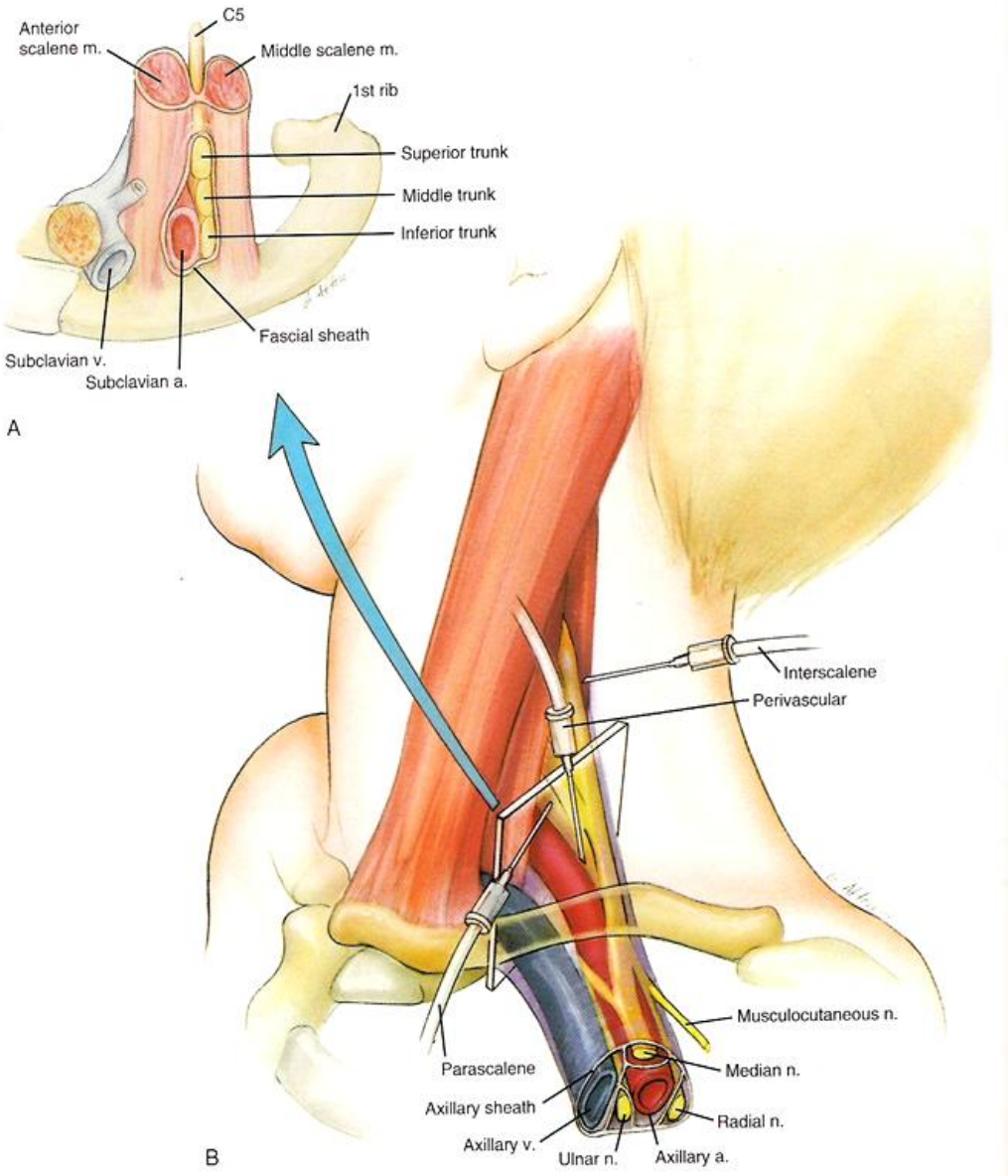
onset of sensory and motor block and duration of analgesia in two groups were compared and development of complications were observed. They found the two groups were comparable indemographic data. The mean onset time of sensory block was 11.64±2.19 minutes in group A and 9.89±1.97 minutes in group B and difference was statistically significant ($p<0.05$). Onset of motor block was 13.32±0.98 minutes in group A and 11.09±1.28minutes in group B and difference was statistically significant ($p<0.05$). There was markedly prolonged duration of analgesia in group-B, 11.87± 0.53 hours compared to group-A, 3.43±0.49 hours. The result was statistically highly significant ($p<0.001$).They concluded that the addition of dexamethasone as an adjuvant to local anaesthetics in brachial plexus block results in significantly early onset and markedly prolonged duration of analgesia without any unwanted effects.

15. Golwala M, Swadia V, Dhimar A, Sridhar N[(14)] studied the effect of dexamethasone as an adjuvant to local anesthetics in supraclavicular brachial plexus block. They concluded that the addition of dexamethasone with local anesthetic prolongs the duration of analgesia. They also found that dexamethasone increases the intensity of motor and sensory blockade.

16. Kopecz DJ, Lacouture PG, WuD, et al[22] studied the dose response and effects of dexamethasone on bupivacaine microcapsules for intercostal blockade in human volunteers and they concluded that dexamethasone microcapsules are well tolerated and inclusion of dexamethasone increased the duration of intercostal block to atleast 96 hours .

17. Ang ET, Gold farb G, Kohn S et al[3] analysed the efficacy of dexamethasone given in epidural for post operative analgesia. They found that addition of dexamethasone with local anesthetics prolongs the post operative analgesia duration.

MATERIALS AND METHODS


C5
Anterior scalene m.
Middle scalene m.
1st rib
Superior trunk
Middle trunk
Inferior trunk
Fascial sheath
Subclavian v.
Subclavian a.
A
Interscalene
Perivascular
Musculocutaneous n.
Parascalene
Median n.
Axillary sheath
Radial n.
Axillary v.
Ulnar n.
Axillary a.
B

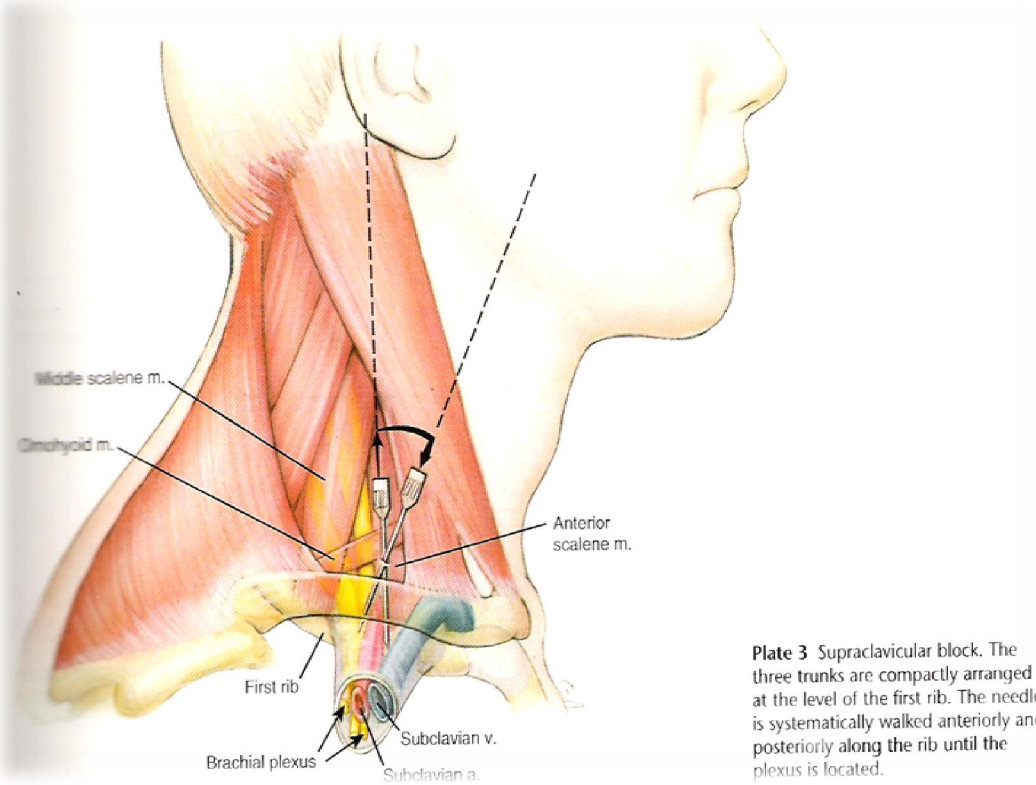


Plate 3 Supraclavicular block. The three trunks are compactly arranged at the level of the first rib. The needle is systematically walked anteriorly and posteriorly along the rib until the plexus is located.

MATERIALS AND METHODS

This study was a prospective randomized comparative study. After receiving the institutional ethical committee approval and informed consent from the patients they were randomly allocated into two groups.

A total number of 60 adult patients of both sexes in the age group of 20 to 60 years belonging to ASA I /II category who were posted for various type of upper limb surgeries in the department of orthopaedics at Government Rajiv Gandhi hospital formed the study group.

Groups :

Group A : 30 patients received 15 ml 2% lignocaine with adrenaline + 15 ml 0.5% bupivacaine with 2 ml of 0.9% normal saline.

Group B : 30 patients received 15 ml 2% lignocaine with adrenaline + 15 ml 0.5% bupivacaine with 8 mg dexamethasone.

INCLUSION CRITERIA :

ASA physical status I / II

Age 20 to 60 years.

Patients undergoing upper limb surgery who have given informed consent.

EXCLUSION CRITERIA :

ASA physical status III / IV

Patients with coagulation abnormalities.

History of allergy to local anesthetics

Patchy or inadequate analgesia

Diabetes

Acid peptic disease

Patient refusal

Patient requiring conversion to GA

Patient not fitting into inclusion criteria

EQUIPMENTS :

Sterile tray

Sterile towel

Sterile swabs

Sponge holding forceps

Povidone iodine solution

10 ml syringe

2ml syringe with 24 G needle

0.5% bupivacaine vial

Freshly prepared 2% lignocaine with adrenaline 1:200000 vial

Dexamethasone 8mg

25 G spinal needle

INTRAOPERATIVE AND POSTOPERATIVE MONITORING :

(1) Heart rate, (2) SPO2, (3) NIBP, (4) ECG

Initially the pre procedure parameters were recorded.ie , heart rate, systolic BP, diastolic BP, MAP, SPO2, ECG. These were taken as baseline values before giving the block. All through the study these parameters were monitored continuously except the NIBP which was recorded intermittently. Postoperatively they were monitored upto regression of motor and sensory blockade. Patients were observed vigilantly for the development of complications.

SUBCLAVIAN PERIVASCULAR TECHNIQUE:

1. IV line was started for all the patients with 18 G I.V cannula after connecting monitors to the patient.

2. Patient was positioned on the table and proper illumination was done at the site of block.

3. For continuous neurological evaluation no sedative drugs were administered preoperatively.

4. Patient placed in supine position with head turned to the side opposite to the side that is to be injected.

5. The arms are at the patient’s side with the hands pointing towards the knee.

6. A rolled towel is placed lengthwise between the shoulders along the spine to give the best exposure to the blocking area.

7. The area is aseptically prepared and draped.

8. The anesthesiologist stands at the head end of the table.

9. The patient is asked to lift the head slightly to bring the clavicular head of sternomastoid into prominence.

10. The index finger is placed lateral to the muscle and the patient is asked to relax. Roll the index finger laterally across the belly of the muscle until the inter scalene groove is palpated.

11. The finger is then moved inferiorly down the groove until the pulse of subclavian artery is palpated.

12. A skin wheal is raised at a point about 2 to 2.5 cm above the midpoint of clavicle with 1 ml of 2% lignocaine by a 24 G needle.

13. The palpation of subclavian artery against the palpating finger is a guide to supraclavicular block.

14. A spinal needle is held between the thumb and index finger, inserted at the point where we raised local anesthetic wheal.

15. The needle is directed towards the ipsilateral nipple, posterolateral to the subclavian artery.

16. Within 2cm a pop off is felt or parasthesia may be elucited, it indicates the needle is inside the sheath and closer to the nerve bundle. Then the local anesthetic solution is given in 5ml increments with frequent aspiration to prevent intravascular injection.

17. Intercostobrachial nerve and medial cutaneous nerve are blocked separately at the axilla anterior to the axillary artery by subcutaneous infiltration of local anesthetic to ensure complete anesthesia of the upper extremity.

18. The needle should not be advanced beyond 2.5cm to avoid the risk of complications. A cough by the patient is a warning that the pleura is being irritated by the needle.

19. After injecting the local anesthetic the block is tested for both sensory and motor blockade and is compared with the contralateral limb.

EVALUATION OF MOTOR BLOCKADE:

Motor block is evaluated by thumb abduction (radial nerve), thumb adduction (ulnar nerve), thumb opposition (median nerve) and flexion of the elbow in supination and pronation of the fore arm (musculocutaneous nerve) for every one minute.

EVALUATION OF SENSORY BLOCKADE:

Sensory blockade is evaluated by pinprick in the area of the above four nerves for every 1 minute. The Hollmen's scale is used in the study for assessing both sensory and motor blockade.

MOTOR BLOCKADE:

0	-	normal muscle power
+	-	slight depression of muscle power as compared with preanesthetic power
++	-	very weak muscular action persisting in muscle.
+++	-	complete blockade with absent muscle power . (onset of motor blockade means 3+)

SENSORY BLOCKADE:

0 - normal sensation for pin prick.

\+ - pin prick felt as sharp pointed but weaker Compared with the same area in other limb

++ - pin prick felt as touch with blunt object

+++ - no perception of pin prick. (onset of sensory blockade means 3+)

Evaluation was carried for every minute after completion of the injection and the time of onset was noted for both sensory and motor blockade. Only patients with complete motor and sensory blockade were included in the study. Once block was complete surgery was allowed to proceed.

Duration of sensory blockade was considered as the time interval between the complete sensory blockade and the onset of regression of sensory blockade – first pain to pin prick.(VAS 3)

Duration of motor blockade was considered as the time interval between the complete motor blockade and onset of regression of motor blockade.

In the postoperative period the regression of motor and sensory blockade was tested for every 30 minutes.

MONITORING :

Monitoring during anesthesia focuses on systemic toxicity of local anesthetic, complications of block like pneumothorax and horners syndrome. Parameters like heart rate, systolic BP, diastolc BP, MAP, SPO2, ECG are recorded preblock and monitored throughout the surgery and in the postoperative period upto the onset of regression of motor and sensory blockade. In the postoperative period pain was assessed using a numerical rating pain score scale (visual analogue scale) where 0 represents no pain,10 represents the worst possible pain.

OBSERVATIONS AND RESULTS

OBSERVATION AND RESULTS

This randomized, prospective, single blinded, case controlled study analyses the effectiveness of dexamethasone as an adjuvant to local anesthetic mixture in providing postoperative analgesia in supraclavicular brachial plexus block.

Results are expressed as mean and standard deviation. All statistical analyses were carried out using SPSS for Windows version 15.0. The *t*-test was used for comparison of quantitative variants. Qualitative variants were compared using the chi-square test. A 'P value' of less than 0.05 was considered statistically significant.

Table 1. Demographic profile: Age

Group	Number	Mean	SD	P value
A	30	37.73	13.385	0.386 N.S
B	30	34.93	11.362	

The mean age of group A is 37.73 and group B is 34.93, the p value is 0.386, it is not statistically significant. Both groups are comparable in terms of age.

Table 2. Demographic profile: Sex

Group	Male		Female		P value
	No	%	No	%	
A	25	83.3	5	16.7	0.739 N.S
B	24	80.0	6	20.0	

The percentage of male patients in group A is 83.3 and in group B is 80.0, the percentage of female patients in group A is 16.7 and in group B is 20.0, p value is 0.739, it is not statistically significant. Both groups are comparable in terms of sex.

Table 3. Demographic profile: BMI

Group	No	Mean	SD	P value
A	30	24.97	1.502	0.288 N.S
B	30	24.60	1.214	

The mean BMI of group A is 24.97 and group B is 24.60, the p value is 0.288, it is not statistically significant. Both groups are comparable in terms of BMI.

Table 4. Demographic profile ASA PS:

Group	ASA I		ASA II		P value
	No	%	No	%	
A	25	83.3	5	16.7	0.718 N.S
B	26	86.7	4	13.3	

In group A 25 patients were ASA I and 5 were ASA II patients. In group B 26 patients were in ASA I and 4 were ASA II patients. The data is statistically not significant ($p>0.05$) and this both groups are comparable in terms of ASA PS Status

Table 5. Onset of motor blockade:

Group	No	Mean	SD	P value
A	30	15.13	0.860	<0.005 SIG
B	30	13.33	0.884	

The mean onset of motor blockade in group A is 15.13 minutes, in group B is 13.33 minutes. Statistical analysis reveal p value <0.005, it is statistically significant.

Table 6. Onset of sensory blockade:

Group	No	Mean	SD	P value
A	30	19.30	0.915	<0.005 SIG
B	30	16.43	0.774	

The mean onset of sensory blockade in group A is 19.30, and in group B is 16.43 minutes. Statistical analysis reveal p value < 0.005, which is statistically significant.

Table 7. Quality of anesthesia:

Group	Quality						P value
	1		2		3		
	No	%	No	%	No	%	
Group A	_		4	13.3	26	86.7	0.161 N.S
Group B	_		1	3.3	29	96.7	

In group A the quality of anesthesia is 3 for 26 patients, 2 for 4 patients and in group B it is 3 for 29 patients and 2 for 1 patient. Statistical analysis shows the p value is 0.161, which is not significant.

Table 8. Duration of surgery:

Group	No	Mean	SD	P value
A	30	152.47	11.530	0.690 N.S
B	30	153.40	15.843	

The mean duration of surgery in group A is 152.47, and in group B is 153.40. statistical analysis show the p value is 0.690. The p value is not statistically significant.

Table 9. Duration of motor blockade:

Group	No	Mean	SD	P value
A	30	207.67	16.219	< 0.005 SIG
B	30	374.93	13.829	

The mean duration of motor blockade in group A is 207.67 minutes, in group B is 374.93 minutes. Statistical analysis show the p value < 0.005, which is statistically significant.

Table 10. Duration of sensory blockade:

Group	No	Mean	SD	P value
A	30	318.77	20.997	<0.005 SIG
B	30	642.33	30.250	

The mean duration of sensory blockade in group A is 318.77 minutes, in group B is 642.33 minutes. Statistical analysis show the p value is <0.005, which is statistically significant.

Table 11. Heart rate:

	Group	No	Mean	SD	P value
Pre block	A	30	79.37	6.615	0.599 NS
	B	30	78.53	4.819	
1 min	A	30	81.67	6.166	0.504 NS
	B	30	82.70	5.742	
5 min	A	30	81.40	6.360	0.561 NS
	B	30	82.40	6.881	
10 min	A	30	79.40	6.621	0.386 NS
	B	30	80.87	6.383	
15 min	A	30	76.97	6.425	0.176 NS
	B	30	79.07	5.407	
30 min	A	30	75.23	5.728	0.195 NS
	B	30	77.13	5.494	
45 min	A	30	74.63	5.762	0.205 NS
	B	30	76.43	5.090	
End	A	30	75.27	4.820	0.935 NS
	B	30	75.37	4.694	

The heart rate is measured pre block and 1 min, 5mins, 10mins,15mins, 30mins, 45mins after the block and at the end of the surgery. Statistical analysis using student t test shows the p value of 0.599, 0.504, 0.561, 0.386, 0.176, 0.195, 0.205, and 0.935 respectively, which are not significant.

Tables 12. Systolic blood pressure:

	Group	No	Mean	SD	P value
Pre block	A	30	126.67	7.232	0.435 NS
	B	30	125.20	7.208	
1 min	A	30	130.07	8.128	0.480 NS
	B	30	128.70	6.691	
5 min	A	30	131.40	6.688	0.694 NS
	B	30	130.77	5.685	
10 min	A	30	128.77	6.426	0.475 NS
	B	30	127.60	6.145	
15 min	A	30	125.23	5.876	0.313 NS
	B	30	123.73	5.539	
30 min	A	30	122.67	6.315	0.367 NS
	B	30	121.30	5.279	
45 min	A	30	120.63	6.014	0.561 NS
	B	30	119.83	4.480	
End	A	30	120.00	4.756	0.895 NS
	B	30	119.83	5.004	

The systolic blood pressure is measured during pre block, 1min, 5mins, 10mins, 15mins, 30mins, 45mins after the block and at the end of the surgery. Statistical analysis using student t test shows the p value of 0.435, 0.480, 0.694, 0.475, 0.313, 0.367, 0.561, and 0.895 respectively, which is not significant.

Table 13. Diastolic blood pressure:

	Group	No	Mean	SD	P value
Pre block	A	30	77.57	6.021	0.769 NS
	B	30	77.13	5.322	
1 min	A	30	80.33	6.326	0.588 NS
	B	30	79.47	5.998	
5 min	A	30	80.77	6.532	0.396 NS
	B	30	79.30	6.742	
10 min	A	30	77.63	6.693	0.405 NS
	B	30	76.23	6.229	
15 min	A	30	74.47	6.511	0.160 NS
	B	30	72.33	4.999	
30 min	A	30	71.73	5.836	0.258 NS
	B	30	70.20	4.483	
45 min	A	30	69.67	5.416	0.849 NS
	B	30	69.43	3.945	
End	A	30	69.03	4.767	0.699 NS
	B	30	69.47	3.821	

Table 14. Complication: Nausea:

Group	Nausea				P value
	YES		NO		
	No	%	No	%	
A	3	10	27	90	0.301 NS
B	1	3.3	29	96.7	

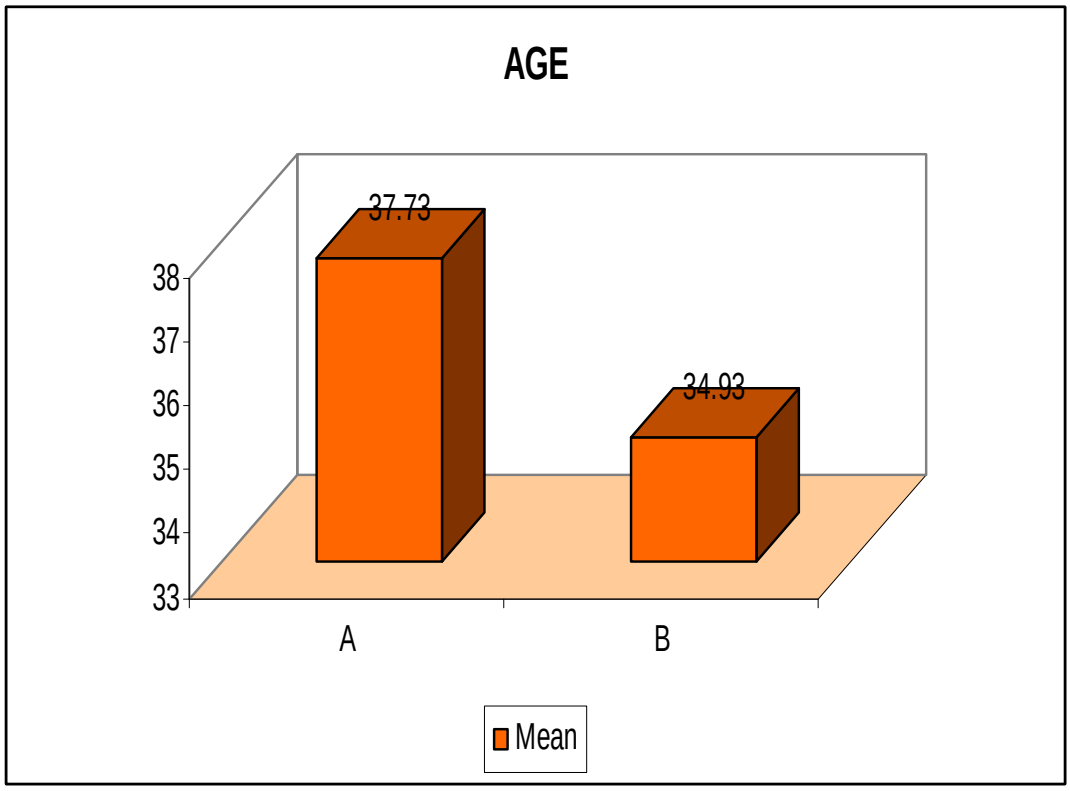
In group A 3 patients complaints of nausea, 27 patients are nausea free, in group B 1 patient complaints of nausea, and 29 patients are free of nausea. Statistical analysis shows the p value of 0.301, which is statistically not significant.

Table 15. Mean Arterial Pressure:

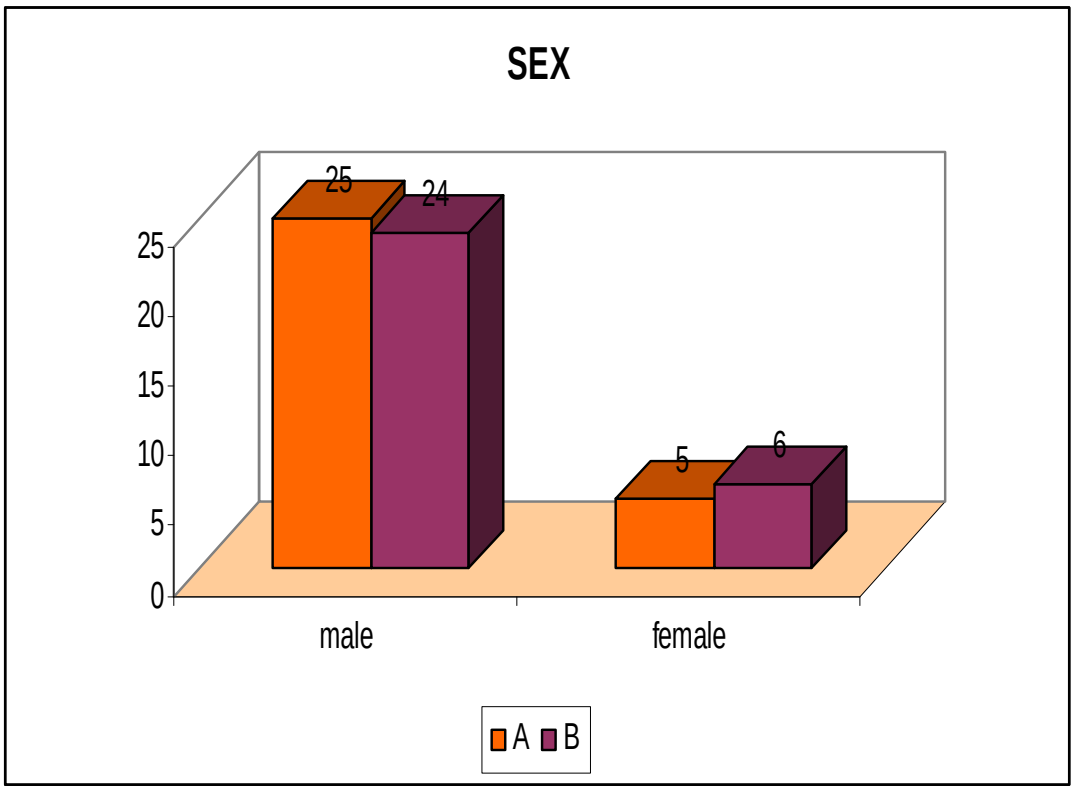
	Group	No	Mean	SD	P value
Pre block	A	30	93.87	6.280	0.633 NS
	B	30	93.13	5.529	
1 min	A	30	96.88	6.733	0.588 NS
	B	30	95.84	5.851	
5min	A	30	97.41	6.150	0.533 NS
	B	30	96.42	6.143	
10 min	A	30	94.51	6.452	0.306 NS
	B	30	92.87	5.802	
15 min	A	30	91.33	6.051	0.164 NS
	B	30	89.33	4.876	
30 min	A	30	88.68	5.760	0.278 NS
	B	30	87.21	4.577	
45 min	A	30	86.62	5.402	0.736 NS
	B	30	86.21	3.913	
End	A	30	85.99	4.543	0.891 NS
	B	30	86.16	4.621	

The diastolic blood pressure and mean arterial pressure are measured during pre block, 1min, 5mins, 10mins, 15mins, 30mins, 45mins, after the block, and at the end of the surgery. The p values are calculated using student t test, which are shown on the table 13 and table 14. The p values are statistically not significant.

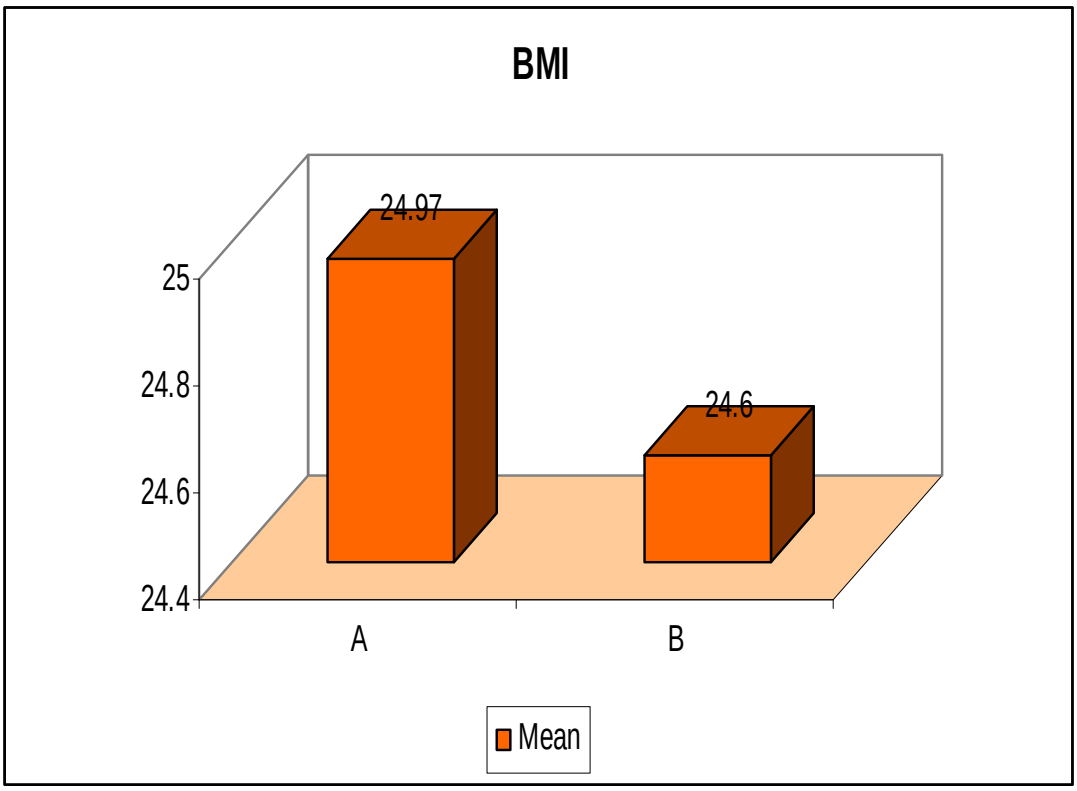
1. AGE



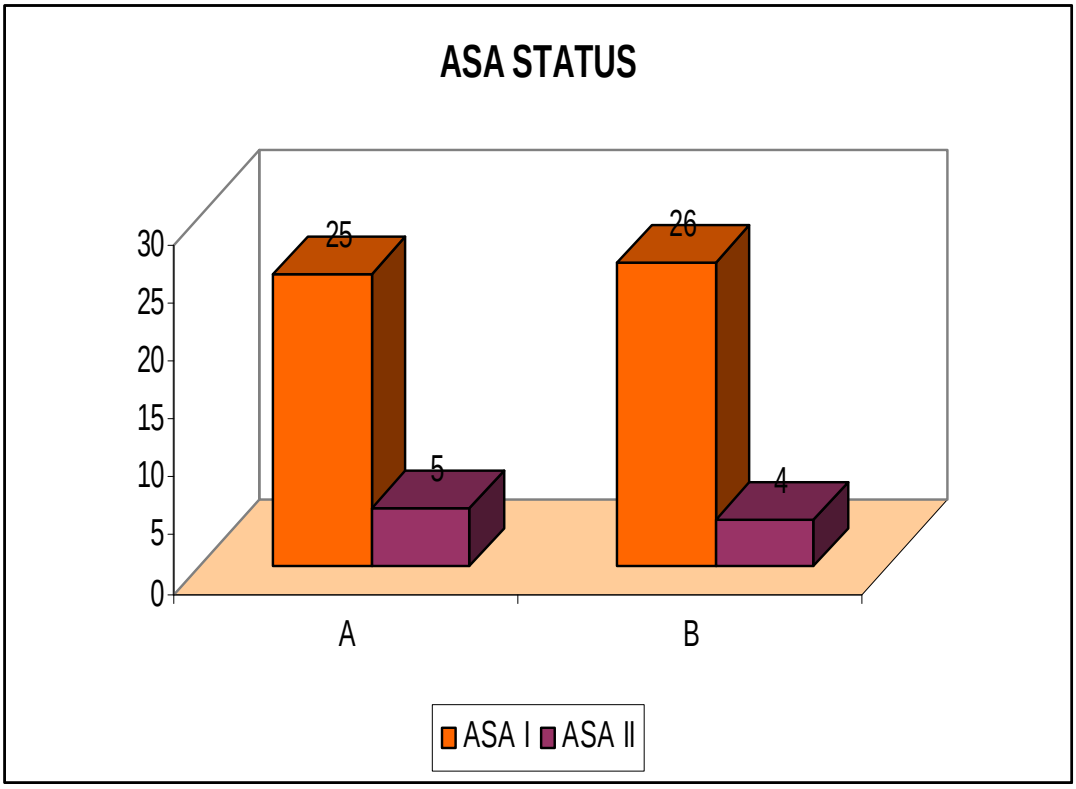
2. SEX



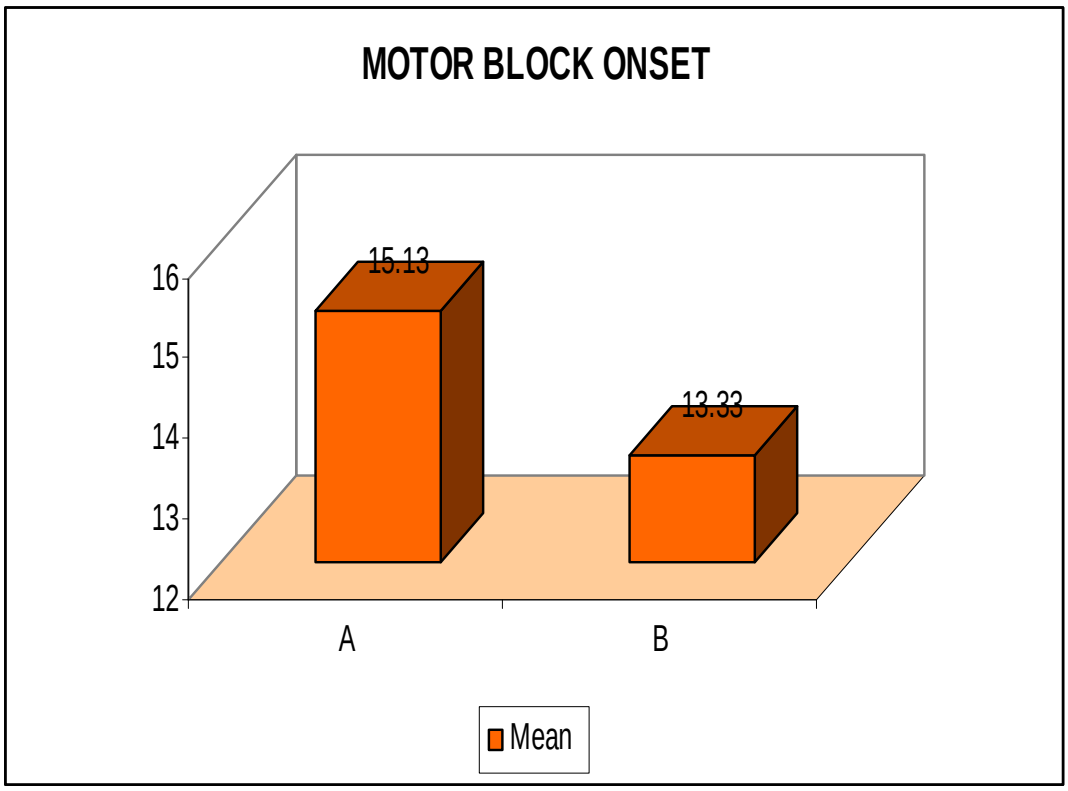
3. BMI



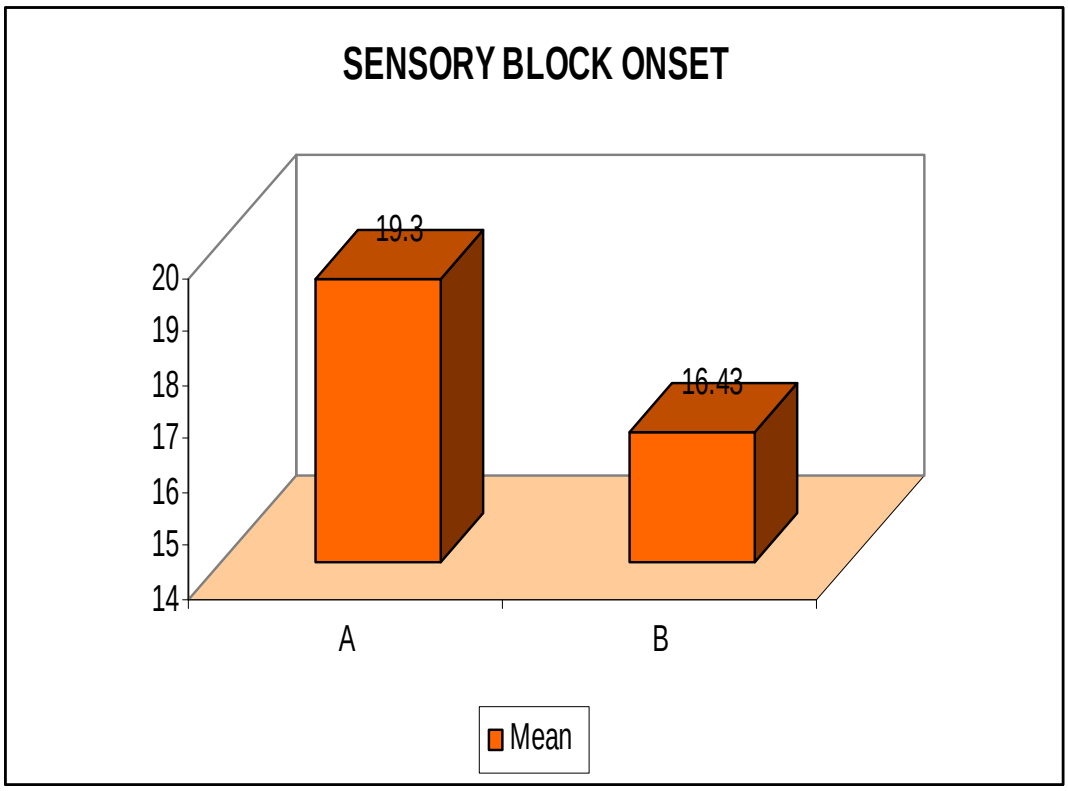
4. ASA STATUS:



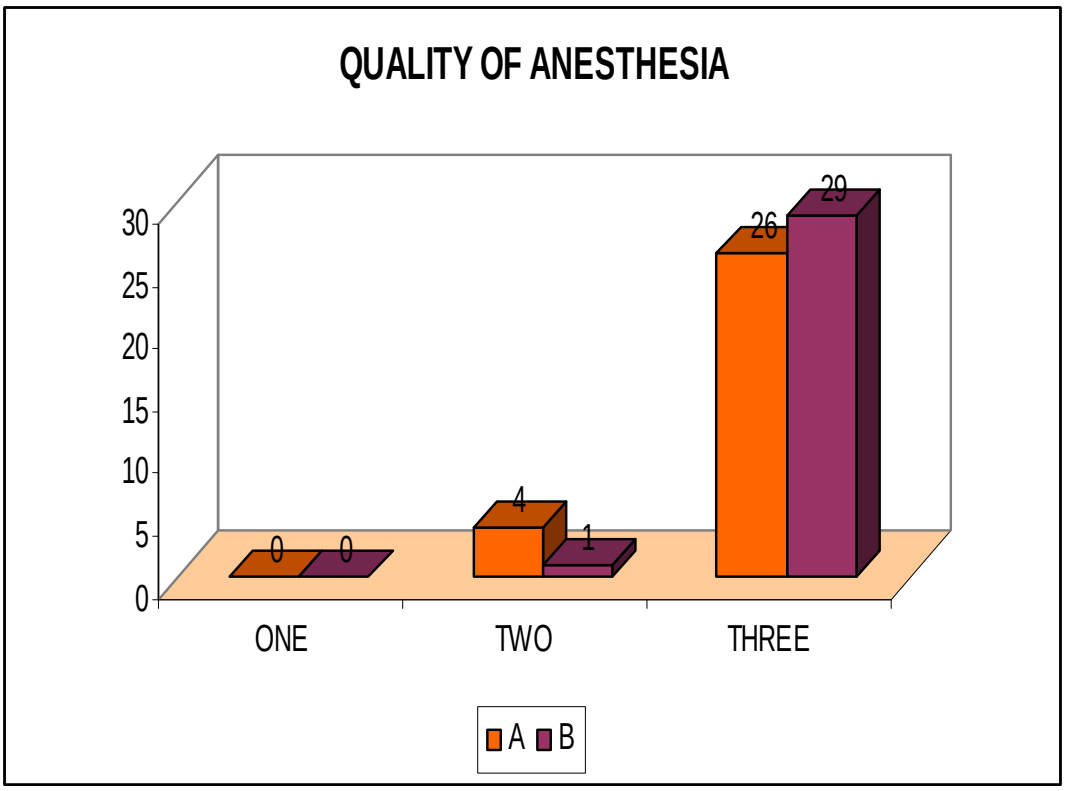
5. MOTOR BLOCK ONSET:



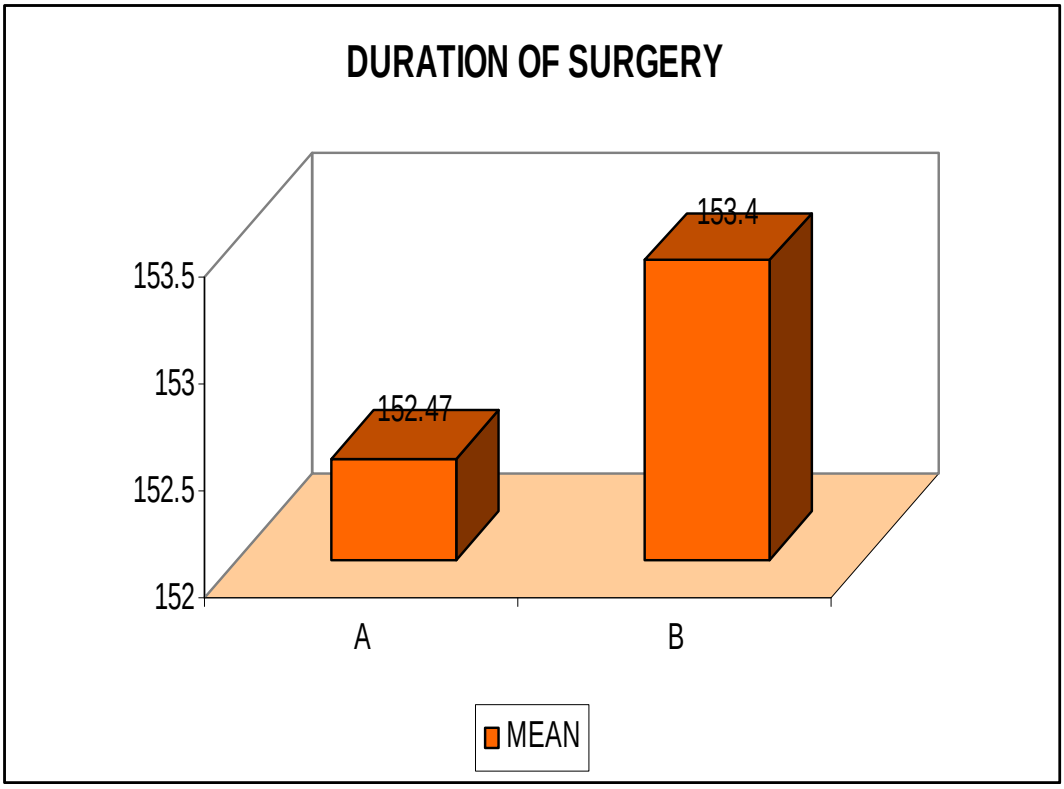
6. SENSORY BLOCK ONSET:



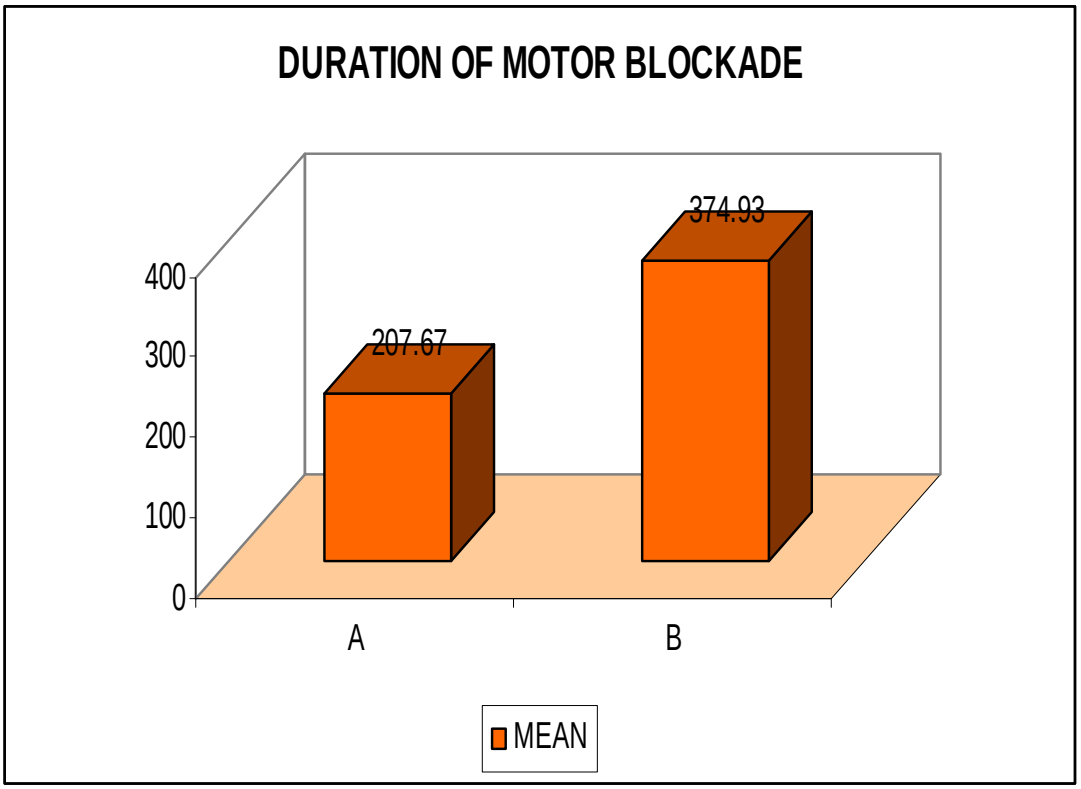
7. QUALITY OF ANESTHESIA:



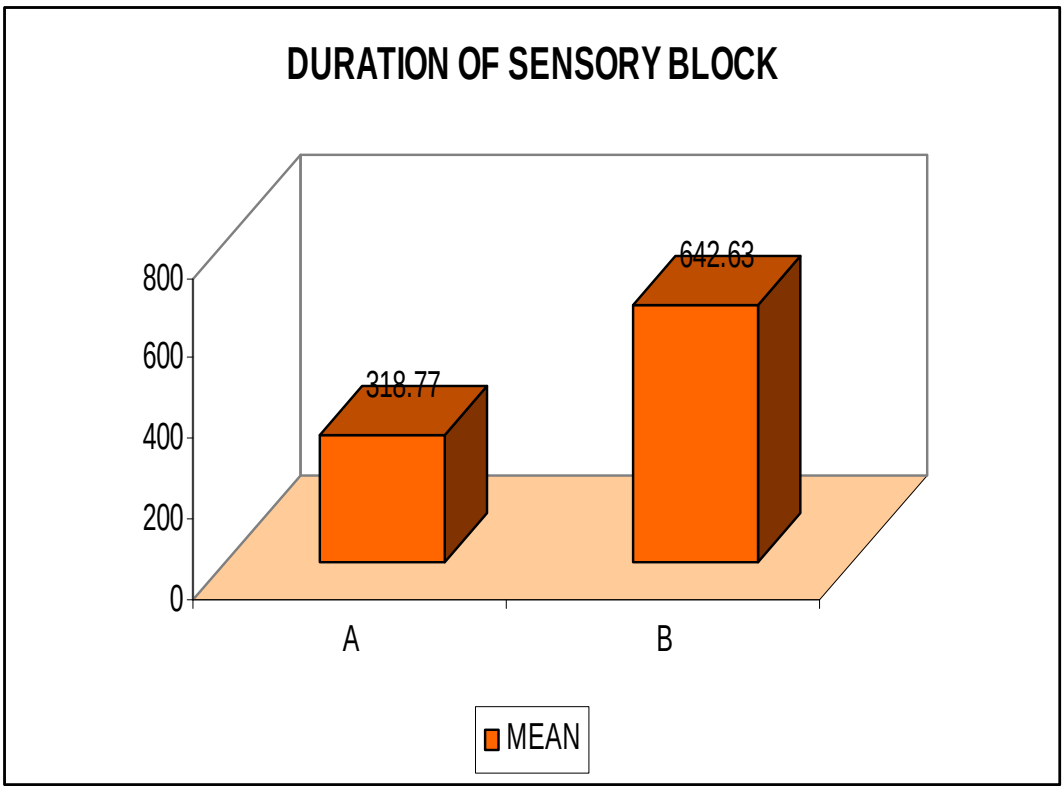
8. DURATION OF SURGERY:



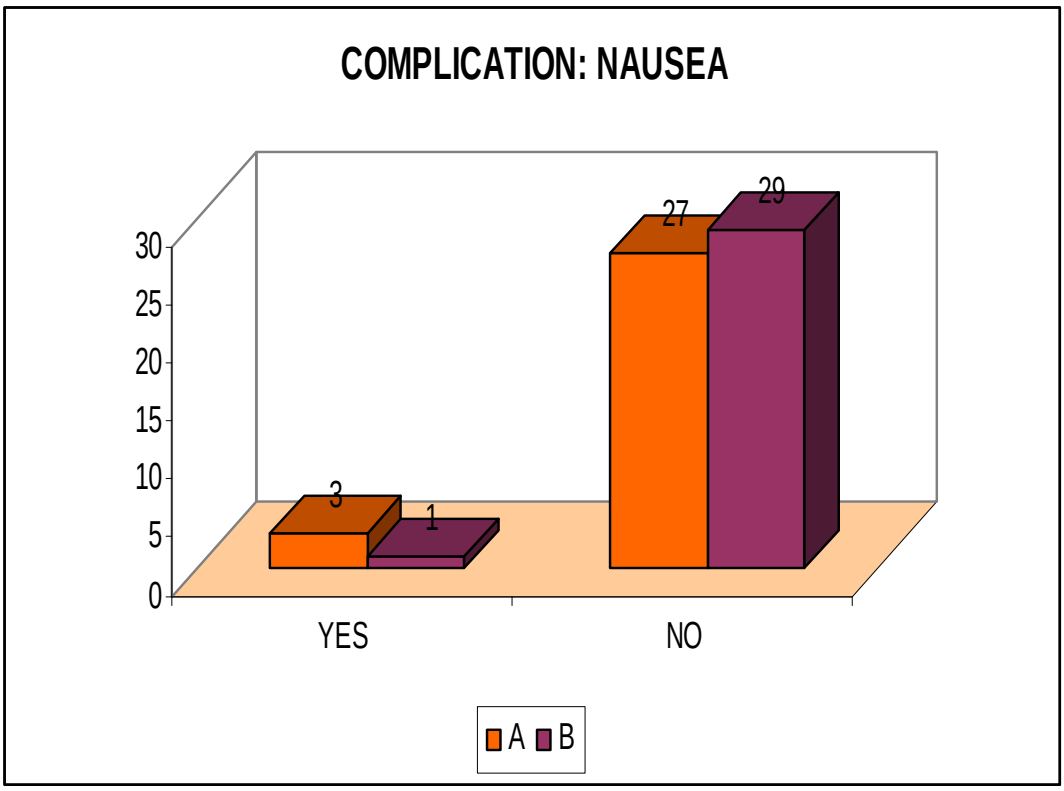
9. DURATION OF MOTOR BLOCKADE:



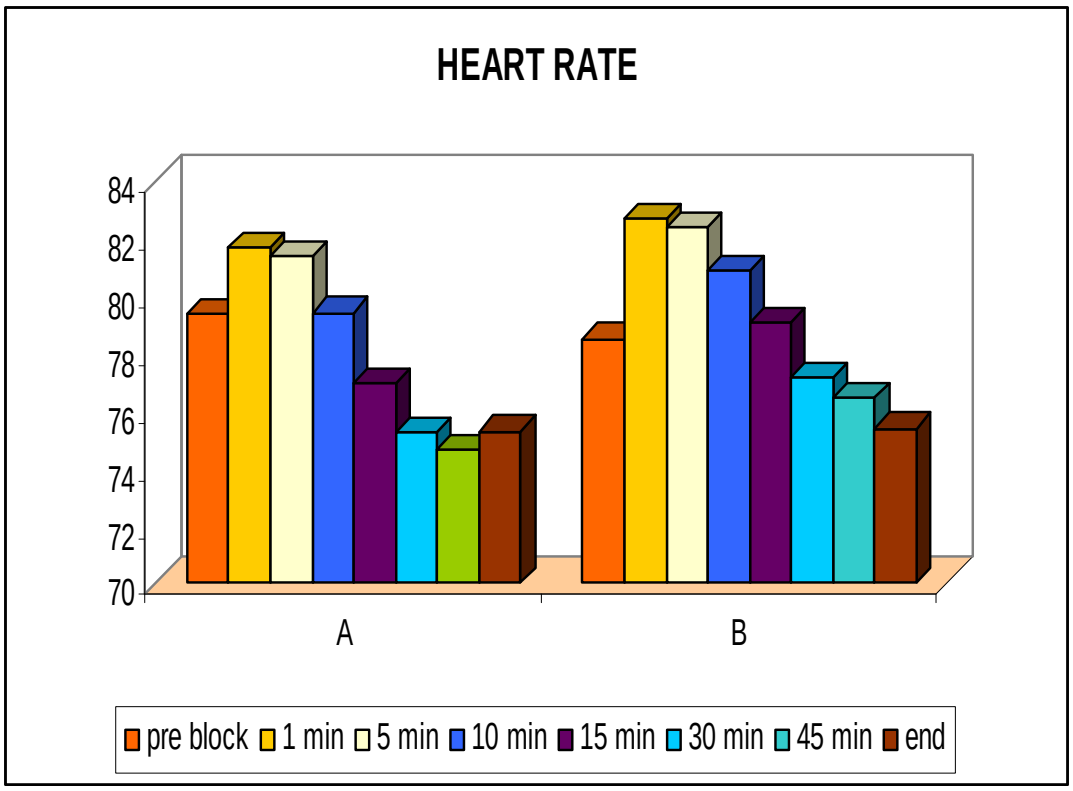
10. DURATION OF SENSORY BLOCKADE:



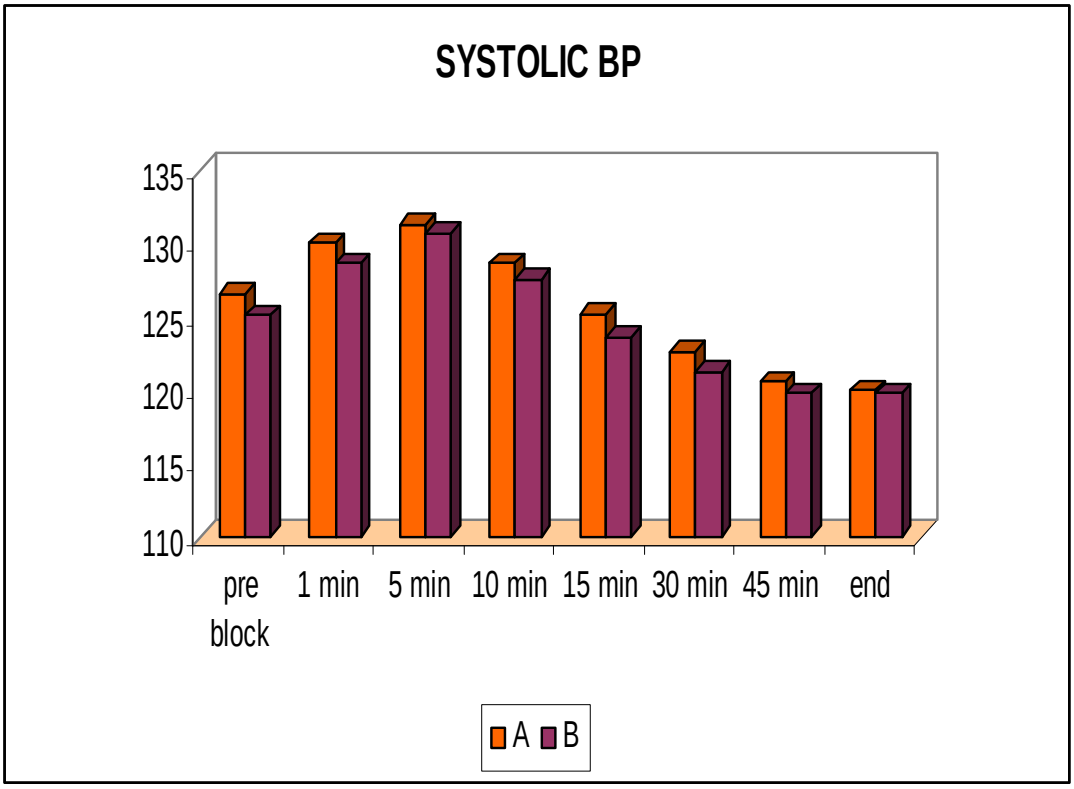
11. COMPLICATIONS:



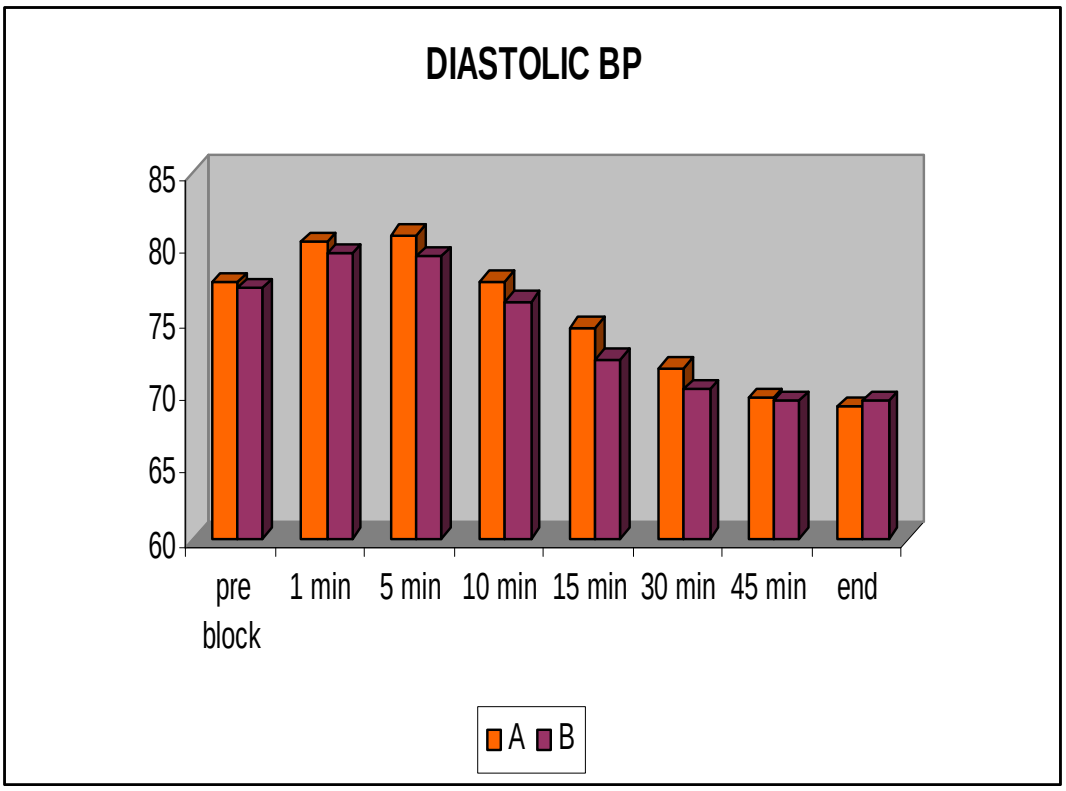
12. HEART RATE:



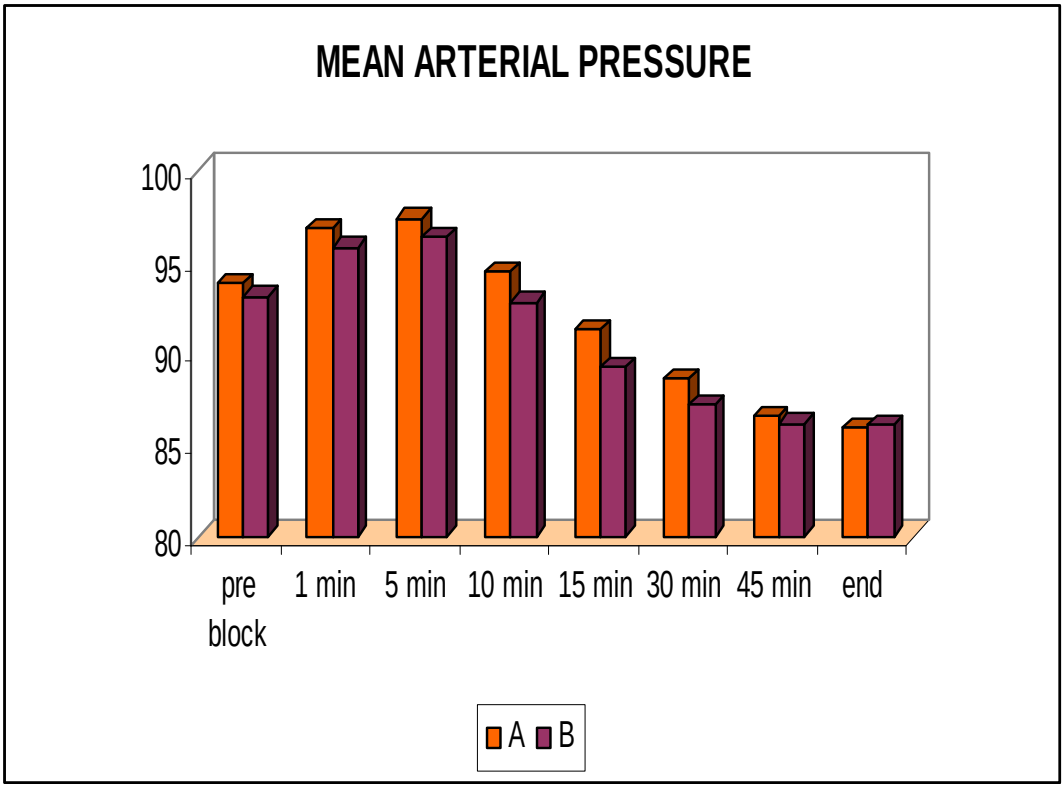
13. SYSTOLIC BLOOD PRESSURE:



14. DIASTOLIC BLOOD PRESSURE:



15. MEAN ARTERIAL PRESSURE:



DISCUSSION

DISCUSSION

Brachial plexus block is an easy and relatively safe procedure for upper limb surgeries. A combination of lignocaine and bupivacaine provided better operating conditions but the duration of analgesia is rarely maintained for more than 4-6 hours. Addition of steroid to local anaesthetics effectively and significantly prolongs the duration of analgesia as well as producing earlier onset of action. Steroids are very potent anti-inflammatory and immunosuppressive agents. Perineural injection of steroid is reported to influence post operative analgesia. Epidural steroids were used for treatment of back pain and sciatica.

Various steroids has been used for this purpose, but dexamethasone a 9 alpha derivative synthetic glucocorticoid is preferred because of its highly potent anti-inflammatory property, about25-30 times as potent as hydrocortisone and without any mineralocorticoid activity. Thus was found to be safer and devoid of potential side effects. Pre-operative administration of dexamethasone by oral and intravenous routes has been shown to reduce overall pain scores and analgesic requirements in the postoperative period without any adverse effects in various dental and general surgical procedures**. (Baxendale et al[(5)], Miralles et al[(26)], Elhakim M et al[(12)]).**

Dexamethasone is also known to reduce post-operative nausea and vomiting. The possible mechanism ofanalgesic and antiemetic actions are due to anti-inflammatory property of Dexamethasone. **(Ahlgren et al[1], Bisgaard T et al[7]).**

It has been also observed that addition of small amount of dexamethasone to local anesthetics prolonged the duration of analgesia after subcutaneous, intercostal block, intra articular, and epidurally. **(Holt et al[16], Kopacz et al[22], Stein et al[38], Ang et al[3]).**

1. In our study mean age in group A is 37.73 and in group B it is 34.93 both groups are comparable in terms of age.

2. Mean value of BMI in group A is 24.97, and in group B is 24.60 p value is not significant, both groups are comparable in terms of BMI.

3. Number of male patients in group A is 25 and in group B is 24, number of female patients in group A is 5 and in group b is 6. Both groups are comparable in terms of sex.

4. Number of patients with ASA I in group A is 25 and in group B is 26 number of patients with ASA II in group A is 5 and in group B is 4. Both groups are comparable in terms of ASA PS status.

5. Quality of anesthesia, in group A surgeons graded 26 cases as grade3 and 4 cases as grade 2. In group B surgeons graded 29 cases as grade 3 and 1 case as grade 2. This finding is not statistically significant.

6. Duration of surgery, in both groups are comparable and not statistically significant.

7. Hemodynamic parameters like heart rate, systolic BP, diastolic BP, mean BP are comparable in both groups, are statistically insignificant.

8. Onset of motor blockade and sensory blockade:

   In our study onset of motor blockade and onset of sensory blockade occurs earlier in dexamethasone group. Our finding is comparable with the study conducted by **Shrestha B.R et al**[35] in 40 patients. They found that complete sensory blockade in dexamethasone group occurs in mean 16.76mins, and complete motor blockade occurs in mean 12.90 mins. The early onset of action might be due to synergistic action of dexamethasone with local anaesthetics on blockage of nerve fibres.

In our study motor blockade occurs earlier than sensory blockade, this finding is comparable with the study conducted by **Winnie in 1977**[44], he described the outer motor fibers are blocked earlier than the sensory fibers which are situated deeper in the plexus at the level of trunk and division. This finding is also comparable with the study conducted by **Shrestha B.R et al**[34]**.**

9. Duration of motor and sensory blockade:

In our study regression of motor blockade occurs earlier than sensory regression, this finding is comparable with the study conducted by **De Jong et al**[9]**.**

The duration of pain relief (postoperative analgesia) was markedly prolonged (mean 642.63) in dexamethasone group, while it was only 318.77mins in control group.This results are similar to findings of study of **Yadav et al**[46]**.**

Ali movafegh et al[27] also stated that dexamethasone prolongs the duration of analgesia in axillary block in 60 patients with lignocaine in their study.

Probable mechanism of action of dexamethasone in prolonging the sensory blockade was postulated by number of studies. **Prithviraj**[31] and **Johanson etal**[18] found that the steroids have the nocioceptive c fibres blocking quality.

In another study **Pennington et al**[29] and **Attardi et al**[4] found that the steroids act by alteration of k+ channel, there by synergistically act with local anesthetics in nerve fibres.

In their study **Ahlgren et al**[1] found that the analgesic action is mediated by anti inflammatory effects of steroids.

Steroids have vasoconstrictory action and this action is attributed that to the analgesia prolonging action similar to the action of adrenaline when added with local anesthetics **Seidenari et al**[33].

Other possibilities are action on corticosteroid receptor in brain after being absorbed from periphery to systemic circulation **(Benzon HT**[6] **and Pieretti et al**[30]**)** suppression of ectopic neuronal discharge **(Devor et al**[10]**).**

In one study after approximately 2000 intrathecal injections of dexamethasone 8mg in 200 patients for treatment of posttramatic visual disturbance **Sugita et al**[39] found no neurological complications.

SUMMARY

SUMMARY

From this prospective, randomised, comparative, single blinded, case control study which evaluated the effectiveness of dexamethasone as an adjuvant to local anesthetic mixture in providing post operative analgesia in supraclavicular brachial plexus block we found that,

1. The demographic profiles like Age, Sex, BMI, ASA status are comparable in both groups.
2. The onset of motor block occurs earlier in dexamethasone group than control group.
3. The onset of sensory block occurs earlier in dexamethasone group than control group.
4. The onset of motor block occurs earlier than sensory block in both groups.
5. The quality of anesthesia, duration of surgery, hemodynamic parameters, complications are comparable in both groups.
6. The duration of motor block is longer in dexamethasone group than control group.
7. The duration of sensory block is longer in dexamethasone group than control group.

CONCLUSION

CONCLUSION

From our study we conclude that addition of dexamethasone 8mg to local anesthetic mixture fastens the onset of action of motor and sensory blockade, and prolongs the duration of motor blockade and analgesia significantly. It may be useful in situations where epinephrine must be used with caution i.e. hypertension and ischemic heart disease.

Further studies are required to elucidate the mechanism of action, determine the optimal dose, and examine the safety profile of dexamethasone before its routine use as perineural adjuant can be advocated.

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MASTER CHART

GROUP A :

S.No.	NAME	AGE	SEX	BMI	ASA	MB	SB
1	RAVINDRAN	47	M	25.2	I	15	19
2	ANJALI	30	F	24.05	I	17	21
3	JAYARAMAN	25	M	24.2	I	14	18
4	ARUL	26	M	25.3	I	16	20
5	SUNDARAMURTI	30	M	24.7	I	15	19
6	VIJAY	26	M	25.7	I	16	21
7	SATHYAMURTI	45	M	24.6	I	14	20
8	PRABHU	25	M	27	I	15	19
9	EDWARD JOHN	58	M	24.2	II	14	18
10	THAYAMMAL	55	F	27.19	II	16	19
11	SANTHAKUMAR	23	M	26.1	I	15	19
12	MUNUSAMI	45	M	23.5	I	14	20
13	KALIDOSS	26	M	24.9	I	15	21
14	SAMPATH	55	M	23.3	II	15	19
15	MURUGAN	39	M	24.2	I	14	18
16	GOPIKANNAN	20	M	24.3	I	14	19
17	DHANALAKSHMI	55	F	25.2	II	16	20
18	DURAIRAJ	55	M	24.1	I	16	20
19	THENDRAL	23	M	25.4	I	16	19
20	JAMIM	20	M	25.3	I	15	19
21	MANIVANNAN	30	M	25.1	I	16	19
22	VASUKI	48	F	29.3	I	15	19
23	MEGARAJ	54	M	26.1	I	14	19
24	SURESH	24	M	22.5	I	14	18
25	ANNAVU	56	M	23.4	II	15	18
26	SUBRAMANI	46	M	24.6	I	15	20
27	VIJAYAN	35	M	22.3	I	16	21
28	DEVAKI	37	F	28	I	16	19
29	HARIBABU	22	M	24.8	I	15	19
30	SUBRAMANI	52	M	24.8	I	16	19

GROUP A :

S.No	Q.O.A	D.O.S	D.O.M.B	D.O.S.B	Nausea	P.T	allergy	palsy	H.syn
1	3	151	221	330	No	No	No	No	No
2	2	154	229	278	No	No	No	No	No
3	3	141	188	308	No	No	No	No	No
4	3	144	199	356	No	No	No	No	No
5	3	156	236	330	No	No	No	No	No
6	2	156	204	299	No	No	No	No	No
7	3	148	193	309	No	No	No	No	No
8	3	158	203	327	Yes	No	No	No	No
9	3	147	222	316	No	No	No	No	No
10	3	157	184	337	No	No	No	No	No
11	3	157	202	321	No	No	No	No	No
12	3	130	210	306	No	No	No	No	No
13	2	152	177	283	No	No	No	No	No
14	3	156	238	340	No	No	No	No	No
15	3	162	227	344	No	No	No	No	No
16	3	159	204	314	Yes	No	No	No	No
17	3	162	217	336	No	No	No	No	No
18	3	145	215	324	No	No	No	No	No
19	3	145	189	333	No	No	No	No	No
20	3	174	209	293	No	No	No	No	No
21	3	156	199	296	No	No	No	No	No
22	3	135	210	304	No	No	No	No	No
23	2	152	207	290	No	No	No	No	No
24	3	172	178	296	No	No	No	No	No
25	3	142	212	335	No	No	No	No	No
26	3	174	205	300	No	No	No	No	No
27	3	134	209	329	No	No	No	No	No
28	3	140	236	352	yes	No	No	No	No
29	3	144	211	343	No	No	No	No	No
30	3	171	196	334	No	No	No	No	No

GROUP A: HEART RATE:

S.No	PRE B	1min	5min	10min	15min	30min	45min	END
1	79	85	89	86	81	77	75	74
2	87	94	93	90	84	80	76	79
3	90	86	82	74	70	67	69	75
4	84	87	83	87	85	84	82	80
5	96	84	77	74	72	68	69	72
6	75	79	77	80	84	83	84	80
7	68	69	74	72	67	65	63	66
8	76	79	84	83	80	79	84	82
9	69	68	70	70	67	68	69	67
10	74	73	77	75	74	75	73	72
11	82	80	86	82	80	76	74	77
12	75	79	84	82	80	77	75	76
13	82	86	84	80	76	74	74	72
14	72	75	77	69	67	68	68	70
15	82	84	80	84	87	83	80	79
16	86	82	87	83	80	76	74	76
17	82	84	83	85	82	80	79	77
18	69	74	67	66	64	67	66	65
19	76	79	83	82	80	76	75	77
20	76	78	74	73	70	69	71	73
21	82	87	83	84	79	76	77	80
22	87	89	91	90	86	84	83	80
23	72	75	70	67	69	67	65	68
24	79	87	86	84	80	81	80	77
25	74	80	76	73	75	77	74	71
26	77	79	83	80	76	75	76	79
27	78	85	84	80	76	77	79	80
28	86	89	90	87	86	82	83	83
29	82	87	83	80	76	75	72	74
30	84	87	85	80	76	71	70	77

GROUP A: SYSTOLIC BLOOD PRESSURE:

S.No	Pre B	5 min	10 min	15 min	20 min	30 min	45 min	End
1	130	136	132	124	122	118	120	124
2	128	129	137	136	130	128	126	122
3	124	128	126	120	118	118	116	119
4	118	110	116	128	120	106	104	108
5	128	136	136	130	127	124	122	120
6	119	128	128	124	118	118	120	124
7	130	136	140	142	137	132	126	118
8	124	126	130	130	127	124	120	117
9	136	139	143	145	137	132	130	124
10	134	138	132	126	120	118	117	112
11	124	120	124	124	122	123	125	126
12	128	130	132	130	127	124	122	118
13	116	120	124	130	132	136	132	127
14	134	139	137	130	130	124	126	120
15	130	132	126	124	119	116	115	112
16	132	136	130	124	122	118	116	119
17	137	139	146	142	137	132	127	122
18	134	137	132	130	127	126	125	120
19	118	120	127	122	118	117	114	114
20	116	124	127	129	125	121	124	125
21	124	126	127	121	118	124	125	126
22	126	134	137	132	124	120	116	112
23	136	139	142	133	130	130	123	120
24	114	119	124	126	127	119	126	125
25	136	139	140	137	132	130	124	120
26	130	136	130	124	120	121	114	124
27	124	128	129	127	126	118	113	119
28	118	116	126	120	124	126	119	124
29	116	124	130	127	120	117	114	121
30	136	138	132	126	121	120	118	118

GROUP A: DIASTOLIC PRESSURE:

S No	Pre B	5 min	10 min	15 min	20 min	30 min	45 min	End
1	81	85	82	76	70	69	70	73
2	76	76	84	82	80	74	74	70
3	72	74	73	67	64	63	62	64
4	70	70	72	75	72	64	64	65
5	73	80	84	80	78	75	71	67
6	72	77	76	72	69	67	70	72
7	84	87	90	92	89	84	80	74
8	73	78	82	81	76	72	69	66
9	87	90	94	92	89	84	74	75
10	85	86	80	74	70	69	65	63
11	77	74	76	75	71	70	72	74
12	79	84	87	83	80	75	72	67
13	70	76	79	80	81	84	83	76
14	82	85	83	80	82	79	75	72
15	76	77	74	74	70	64	64	60
16	84	87	82	77	73	70	67	70
17	89	92	95	93	84	80	75	74
18	85	86	83	81	76	74	72	70
19	73	75	76	71	69	67	64	62
20	70	72	73	75	73	70	72	77
21	74	76	75	70	70	70	77	74
22	70	77	76	72	67	66	62	60
23	84	87	90	75	72	70	65	63
24	72	74	75	73	74	70	72	72
25	85	89	91	86	81	76	72	70
26	80	84	74	69	63	65	64	70
27	75	77	74	73	72	70	64	66
28	72	70	78	72	70	70	64	67
29	73	78	82	79	73	69	65	71
30	84	87	83	80	76	72	70	67

GROUP A : MEAN ARTERIAL PRESSURE:

S No	Pre B	5 min	10 min	15 min	20 min	30 min	45 min	End
1	97.3	102	98.6	92	87.3	85	86.6	90
2	93.3	93.6	101.6	100	96.6	92	91.3	87.3
3	89.3	92	90.6	84.6	82	81.3	80	82.3
4	86	83.3	86.6	92.6	88	78	77.3	79.3
5	90.3	98.6	101.3	96.6	94.3	91.3	88	84.6
6	87.6	94	93.3	89.3	85.3	84	86.6	89.3
7	99.3	103.3	106.6	108.6	104	100	95.3	88.6
8	90	94	98	97.3	93	89.3	86	83
9	103.3	106.3	110.3	109.6	105	100	92.6	91.3
10	101.3	103.3	97.3	91.3	86.6	85.3	82.3	79.3
11	92.6	89.3	92	91.3	88	87.6	89.6	91.3
12	95.3	99.3	102	98.6	95.6	91.3	88.6	84
13	85.3	90.6	94	96.6	98	101.3	99.3	93
14	99.3	103	101	96.6	98	94	92	88
15	94	95.3	91.3	90.6	86.3	81.3	81	77.3
16	100	103.3	98	92.6	89.3	86	83.3	86.3
17	105	107.6	112	109.3	101.6	97.3	92.3	90
18	101.3	103	99.3	97.3	93	91.3	89.6	86.6
19	88	90	93	88	85.3	83.6	80.6	79.3
20	85.3	89.3	91	93	90.3	87	89.3	93
21	90.6	92.6	92.3	87	86	88	93	91.3
22	88.6	96	96.3	92	86	84	80	77.3
23	101.3	104.3	101.3	94.3	91.3	90	84.3	82
24	86	89	91.3	90.6	91.6	86.3	90	89.6
25	102	105.6	107.3	103	98	94	89.3	86.6
26	96.6	101.3	92.6	87.3	82	83.6	80.6	88
27	91.3	94	92.3	91	90	86	80.3	83.6
28	87.3	85.3	94	88	88	88.6	82.3	86
29	87.3	93.3	98	95	88.6	85	81.3	87.6
30	101.3	104	99.3	91.3	91	88	86	84

MASTER CHART:

GROUP B :

S.No	NAME	AGE	SEX	BMI	ASA	MB	SB
1	CHINASAMY	56	M	24.4	II	12	15
2	MUTHUKRISHNAN	56	M	24.8	I	13	17
3	KANIAPPAN	50	M	24.6	I	13	16
4	PARTHASARATI	46	M	25.3	I	14	17
5	DANAPAL	34	M	26.3	I	15	16
6	ELAMARI	50	F	26.4	II	12	16
7	MOORTHI	25	M	23	I	14	17
8	MAHESWARI	40	F	23.9	I	13	16
9	KARTHIK	32	M	22.7	I	15	18
10	MANIVEL	33	M	23.2	I	12	16
11	PARVANDADAS	21	M	25.19	I	13	18
12	MANJULA	24	F	24.5	I	13	16
13	SELVARAJ	36	M	25.5	I	14	16
14	BACKYARAJ	28	M	24.5	I	13	16
15	ELANGO	26	M	23.7	I	14	17
16	GOPAL	35	M	24.4	I	13	16
17	KARUPASAMI	58	M	24.6	II	12	17
18	RAVISHANKAR	22	M	25	I	13	16
19	SUMITHRA	24	F	25.9	I	14	15
20	LOKESH	35	M	24.2	I	14	16
21	KRISNAMOORTI	35	M	23	I	13	16
22	SHANMUGAM	40	M	22.3	I	13	17
23	NAVEEN	22	M	26.5	I	14	18
24	JODHA	29	M	24.1	I	15	17
25	SATISH KUMAR	40	M	24.8	I	13	16
26	MALARVIZHI	48	F	25.7	II	14	17
27	VENKATESH	33	M	26.1	I	14	16
28	TAMILMANI	20	M	24.8	I	13	17
29	RAGUPATI	21	M	22.3	I	12	16
30	POORI	29	F	26.3	I	13	16

GROUP B :

SNO	Q.O.A	D.O.S	D.O.M.B	D.O.S.B	Nausea	P.T	Allergy	Palsy	H.synd
1	3	174	368	652	No	No	No	No	No
2	3	182	362	612	No	No	No	No	No
3	3	163	359	653	No	No	No	No	No
4	3	152	382	646	No	No	No	No	No
5	3	138	378	665	No	No	No	No	No
6	3	128	374	698	No	No	No	No	No
7	3	129	369	633	No	No	No	No	No
8	3	169	377	603	No	No	No	No	No
9	3	163	386	640	No	No	No	No	No
10	3	151	357	702	No	No	No	No	No
11	3	173	399	685	No	No	No	No	No
12	3	156	396	671	No	No	No	No	No
13	3	161	391	660	No	No	No	No	No
14	3	155	367	641	No	No	No	No	No
15	3	168	373	637	No	No	No	No	No
16	3	151	380	644	No	No	No	No	No
17	3	138	397	658	No	No	No	No	No
18	3	148	398	612	No	No	No	No	No
19	2	160	370	597	No	No	No	No	No
20	3	172	377	605	No	No	No	No	No
21	3	153	363	647	No	No	No	No	No
22	3	145	355	635	No	No	No	No	No
23	3	149	364	599	No	No	No	No	No
24	3	166	371	596	Yes	No	No	No	No
25	3	154	394	703	No	No	No	No	No
26	3	140	390	644	No	No	No	No	No
27	3	135	375	653	No	No	No	No	No
28	3	123	363	628	No	No	No	No	No
29	3	138	353	613	No	No	No	No	No
30	3	183	360	647	No	No	No	No	No

GROUP B: HEART RATE:

S No	PRE B	1min	5min	10min	15min	30min	45min	End
1	72	72	73	70	67	62	68	67
2	74	73	70	67	69	70	71	73
3	69	74	72	70	73	74	72	70
4	75	79	73	74	76	72	77	71
5	80	83	82	80	76	75	72	76
6	69	72	68	70	71	73	70	67
7	77	84	87	89	84	82	80	77
8	74	87	89	90	83	80	81	77
9	80	86	83	87	80	76	79	75
10	82	89	92	87	86	80	77	76
11	84	86	89	87	83	80	82	81
12	87	92	94	89	86	82	83	84
13	79	76	72	77	74	75	73	72
14	86	82	87	86	84	83	82	81
15	82	87	86	82	80	76	74	72
16	76	79	82	80	77	75	76	78
17	69	74	77	75	72	67	66	65
18	80	87	89	86	85	87	87	83
19	79	84	86	83	85	83	80	76
20	77	82	80	81	83	82	79	78
21	82	87	85	83	80	79	77	74
22	80	83	80	79	76	73	75	76
23	82	87	86	87	84	82	83	80
24	87	89	89	86	83	82	79	78
25	77	76	79	74	75	73	70	70
26	79	86	83	82	80	81	76	75
27	80	83	80	81	79	77	80	78
28	79	89	90	77	74	72	70	75
29	80	87	86	83	85	83	80	81
30	79	86	83	84	82	78	74	75

GROUP B: SYSTOLIC BLOOD PRESSURE:

S. No	PRE B	5 min	10 min	15 min	20 min	30 min	45 min	End
1	132	135	139	132	130	126	130	132
2	130	132	135	130	126	124	120	118
3	134	136	137	128	122	118	116	119
4	136	132	130	124	126	119	117	120
5	118	116	124	120	118	116	112	107
6	130	128	136	132	124	122	124	120
7	126	129	134	132	127	120	118	117
8	130	136	139	132	126	122	116	115
9	116	126	123	121	117	116	119	121
10	108	112	126	122	115	114	117	119
11	126	129	126	120	119	121	123	127
12	128	132	130	125	120	116	115	118
13	112	118	136	132	124	122	118	116
14	118	134	133	126	119	117	119	124
15	124	130	125	120	118	120	124	126
16	127	130	128	126	118	116	118	121
17	136	142	144	147	136	132	130	124
18	118	126	127	122	118	116	118	124
19	120	118	126	128	122	129	126	118
20	126	127	129	132	127	122	118	118
21	132	136	132	130	134	130	126	120
22	130	132	127	122	118	115	117	119
23	118	126	125	124	121	122	118	118
24	128	127	130	126	122	119	122	126
25	124	128	124	127	126	118	116	112
26	135	138	143	142	136	130	124	123
27	130	132	130	124	122	116	114	114
28	126	124	129	124	129	132	124	126
29	120	126	129	130	126	124	118	117
30	118	124	127	128	126	125	118	116

GROUP B : DIASTOLIC BLOOD PRESSYRE:

S. No	PRE B	5 min	10 min	15 min	20 min	30 min	45 min	End
1	86	89	92	84	80	77	76	72
2	83	85	80	82	76	74	73	70
3	87	92	97	82	74	69	67	69
4	83	80	81	77	72	70	67	71
5	70	74	75	70	67	65	63	64
6	85	83	84	83	76	72	70	72
7	72	75	79	74	72	66	65	63
8	76	84	87	84	75	67	70	67
9	73	75	70	67	65	66	70	72
10	72	74	78	72	70	67	69	67
11	75	73	70	70	67	70	72	74
12	80	84	82	74	66	67	67	69
13	76	77	79	80	75	72	70	67
14	74	79	74	70	67	66	70	71
15	75	77	73	64	65	70	72	73
16	82	84	82	80	74	71	73	70
17	88	92	90	91	87	82	80	75
18	75	79	75	70	67	65	66	70
19	74	72	72	75	70	73	70	70
20	73	72	73	79	75	70	67	69
21	79	84	83	79	80	76	72	68
22	76	79	74	70	67	62	63	67
23	74	77	73	70	70	69	67	68
24	75	73	78	74	71	70	72	76
25	70	75	72	74	73	67	62	60
26	83	87	89	82	74	72	70	71
27	80	84	83	74	70	67	66	63
28	72	70	76	72	75	80	75	77
29	70	76	78	82	75	72	69	70
30	76	79	80	82	75	72	70	69

GROUP B: MEAN ARTERIAL PRESSURE:

S. No	PRE B	5 min	10 min	15 min	20 min	30 min	45 min	End
1	101.3	104.3	107.6	100	96.6	93.3	94	92
2	98.6	100.6	98.3	98	92.6	90.6	88.6	86
3	102.6	106.6	110.3	97.3	90	85.3	83.3	85.6
4	100.6	97.3	97.3	92.6	90	86.3	83.6	87.3
5	86	88	91.3	86.6	84	82	79.3	78.3
6	100	98	101.3	99.3	92	88.6	88	88
7	90	93	97.3	93.3	90.3	84	82.6	81
8	94	101.3	104.3	100	92	85.3	85.3	83
9	87.3	92	87.6	85	82.3	83	86.3	88.3
10	84	86.6	94	88.6	85	82.6	85	74.3
11	92	91.6	88.6	86.6	84.3	87	89	91.6
12	96	100	98	91	84	83.3	83	85.3
13	88	90.6	98	97.3	91.3	88.6	86	83.3
14	88.6	97.3	93.6	88.6	84.3	83	86.3	88.6
15	91.3	94.6	90.3	82.6	82.6	86.6	89.3	90.6
16	97	99.3	97.3	95.3	88.6	86	88	87
17	104	108.6	108	109.6	103.3	98.6	96.6	91.3
18	89.3	94.6	92.3	87.3	84	82	83.3	88
19	89.3	87.3	90	92.6	87.3	91.6	88.6	86
20	90.6	90.3	91.6	96.6	92.3	87.3	84	89.3
21	96.6	101.3	99.3	96	98	94	90	89.3
22	94	96.6	91.6	87.3	84	79.6	81	84.3
23	88.6	93.3	90.3	88	87	86.6	84	84.6
24	92.6	91	95.3	91.3	88	86.3	88.6	92.6
25	88	92.6	89.3	91.6	90.6	84	80	77.3
26	100.6	104	107	88.6	91.3	91.3	88	88.3
27	96.6	100	98.6	90.6	87.3	83.3	82	80
28	90	88	93.6	89.3	93	97.3	91.3	93.3
29	86.6	92.6	95	98	92	89.3	85.3	85.6
30	90	94	95.6	97.3	92	89.6	86	84.6

PROFORMA

PROSPECTIVE, RANDOMISED, SINGLE BLINDED, CONTROLLED STUDY ON THE EFFECTIVENESS OF DEXAMETHASONE AS AN ADJUVANT TO LOCAL ANESTHETIC MIXTURE IN PROVIDING POSTOPERATIVE ANALGESIA FOR SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK

NAME : AGE : SEX : I.P.No :

DIAGNOSIS : SURGERY PLANNED:

Group A/B Dose of Bupivacaine :

Dose of Dexamethasone:

PREOPERATIVE ASSESSMENT:

HISTORY:

CO-MORBID ILLNESS & TREATMENT DETAILS:

EFFORT TOLERANCE- _____ METS

H/O PREVIOUS SURGERY :

H/O DRUG ALLERGY :

GENERAL EXAMINATION:

HEIGHT: WEIGHT:

ANAEMIA- JAUNDICE- PEDAL EDEMA - AIRWAY-

PULSE- BP- CVS- RS-

INVESTIGATIONS:

Hb : BT: CT: BLOOD GROUPING & TYPING:

BLOOD SUGAR: UREA: CREATININE:

ECG: CXR:

SUPRACLAVICULAR BLOCK :

Perivascular technique

PARAMETERS TO BE OBSERVED :

Time to achieve complete sensory blockade

Time to achieve complete motor blockade

Duration of analgesia

Onset of pain in the postoperative period

Onset of motor regression

Quality of analgesia using VAS score

INTRA OP VITAL PARAMETERS:

TIME	PR	SBP	DBP	SpO_2	RR	Side Effects
Base line						
5 min						
10 min						
15 min						
20 min						
25 min						
30 min						
35 min						
40 min						
45 min						
50 min						
55 min						
60 min						
65 min						
70 min						
75 min						
80 min						
85 min						
90 min						

SIDE EFFECTS :

Side effects	
Nausea / vomiting	
Pruritis	
Hypotension	
Bradycardia	

INTRA OP EVENTS:

IV FLUIDS :

CONVERSION TO GA :

QUALITY OF SURGICAL ANAESTHESIA :

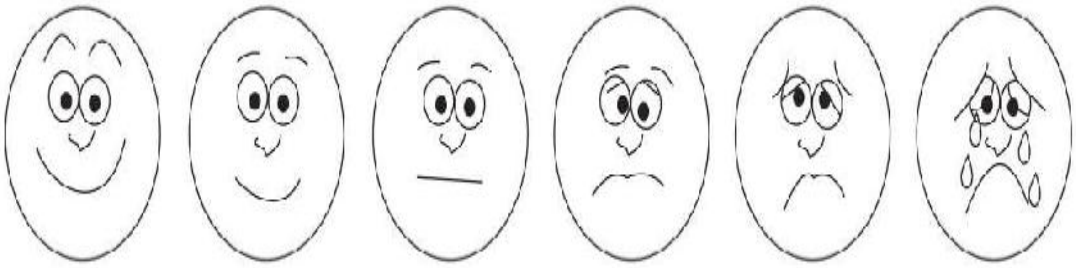
EXCELLENT	GOOD	POOR

PAIN SCORE : (VERBAL RATING SCALE)

ENDING TIME : Onset of pain and motor regression in the postoperative period

VAS SCALE

Visual Analog Scale (VAS)



Very Happy, no hurt.	Hurts just a little bit.	Hurts a little more.	Hurts even more.	Hurts a Whole lot.	Hurts as Much as you can imagine

0 1 2 3 4 5 6 7 8 9 10

No pain Severe pain